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ORIGINAL ARTICLES

THE POSSIBILITIES OF INSULIN IN GENERAL PRACTICE*

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INTRODUCTION

In this country there are at present approximately a million persons ill with diabetes and their number is increasing rather than diminishing. While many diabetics will go to special clinics for treatment, the majority will depend upon their family doctor for advice and guidance. Insulin is now a well established aid in the treatment of certain cases. The general practitioner, therefore, must have a clear idea of how and when to use it.

THE TYPE OF CASE NEEDING INSULIN

It is not a difficult matter to decide categorically whether a given case of diabetes does or does not need insulin. As a rule thin undernourished diabetics of any age, most diabetics under forty, all diabetics with acidosis or a complicating infection, and all diabetics forced to undergo a surgical operation need insulin, while elderly or obese diabetics do not. There are two simple pieces of apparatus which are of the greatest help in quickly separating those patients who may need insulin from those who may not. The first is a pair of ordinary scales to which is attached an instrument for measuring height, and the second is a table† showing the average weight of a person of any given height or age.

Every diabetic patient should have his age, height and weight recorded, and should be asked his maximum weight and when he weighed it because from such simple data alone he gives a fair idea as to whether or not he needs insulin.

In Table 2 is recorded the height-weight-age data on twenty-four recent ambulatory cases. Sixteen of these (67%) had been definitely overweight at some period in their lives, nine (38%) were overweight at present, seventeen (71%) were more than forty years old, and only nine (38%) appeared to need insulin immediately.

This table illustrates certain well known clinical features in regard to diabetes which are always impressive: Diabetes tends to be a mild disease more commonly found in elderly people than in young people, it occurs more frequently in obese people than in thin, and only about one-third of the cases ordinarily seen need insulin. The table brings out, also, the commonly accepted reasons for using insulin in cases without infection or coma. Case 3, a woman, needed insulin because she had a complicating gall-bladder disease which required operation. Two small doses of insulin made her urine sugar-free. The gall bladder was drained under ether anesthesia and a large gall stone was removed. Insulin readily controlled the glycosuria which developed for a few days after operation. Soon, however, the patient regained a high tolerance and was discharged from the hospital on a liberal diet without the bother of daily injections. Case 9, a woman, needed insulin because she was extremely emaciated. Case 10, a man, because he was one of Dr. Joslin's "pedigreed" cases of fourteen years' standing, a little too thin and weak with his present tolerance. He seemed to deserve the drug if any one did after waiting faithfully so long for it. Cases 8 and 16 needed insulin because they were losing weight and strength rapidly despite conscientious attempts at diet. Cases 20, 21, 23 and 24 needed insulin because they were young and evidently severe cases. On the other hand, of the younger patients case 19, a woman 36 years old, did not need insulin because she was holding normal weight and strength without trouble or glycosuria after having lost over thirty pounds of excess flesh, and case 22, another woman 30 years old, because she was more than thirty pounds overweight so that insulin would have only been a handicap to her in her efforts to grow thin.

THE QUESTION OF HOSPITAL FOR PATIENTS WHO ARE TO RECEIVE INSULIN

If it seems best to advise a patient to take insulin the problem of how to give it arises. At present there is a tendency to advise a period of hospital life for all patients who are to re-

*Read before the Central New York Medical Society at Rochester, N. Y., October 23, 1924.

†Dr. Joslin has constructed a card of convenient size which gives these figures.

ceive insulin. Most hospitals keep their diabetic patients under observation for almost three weeks although Dr. Joslin, in his clinic, finds an average stay of ten days sufficient. The patient goes to the hospital for several reasons: in order that the urine may be made sugar free, in order that an adequate diet without glycosuria may be established, in order that the patient may learn how to prepare his diet, how to test his own urine for sugar and how to administer insulin, and finally in order that he may learn something about the natural course and complications of diabetes. The amount of insulin required by each case varies. Most of the Brigham Hospital cases are taking two doses a day of five or ten units; rare cases of maximum severity are taking as much as forty units three times a day. Patients who seem to need insulin should be told at the outset that the drug is administered hypodermically, that it costs a cent a unit, that it does not cure diabetes and that it must be taken indefinitely in view of our present knowledge. Certain cases require less insulin as time goes on: others require more. Nobody enjoys taking it.

The period of hospitalization should be shortened as much as possible because the number of diabetic patients needing hospital treatment is far larger than the supply of hospital beds for the purpose, and because hospital life is expen-

TABLE 1

DR. JOSLIN'S HEIGHT-WEIGHT TABLE

Average Weight of Women in Pounds with Clothes

	Feet and inches with shoes																
Age	4–8	4–9	4–10	4–11	5–0	5–1	5–2	5–3	5–4	5–5	5–6	5–7	5–8	5–9	5–10	5–11	6–0
16	102	104	106	108	109	111	114	117	120	124	128	132	136	139	143	148	153
18	104	106	108	110	112	114	117	120	123	126	130	134	138	141	145	150	155
20	106	108	110	112	114	116	119	122	125	128	132	136	140	143	147	151	156
22	107	109	111	113	115	117	120	123	126	129	133	137	141	145	149	153	157
24	109	111	113	115	117	119	121	124	127	130	134	138	142	146	150	154	158
26	110	112	114	116	118	120	122	125	128	131	135	139	143	147	151	155	159
28	111	113	115	117	119	121	123	126	130	133	137	141	145	149	153	156	160
30	112	114	116	118	120	122	124	127	131	134	138	142	146	150	154	157	161
32	113	115	117	119	121	123	125	128	132	136	140	144	148	152	155	158	162
34	115	117	119	121	123	125	127	130	134	138	142	146	150	154	157	160	163
36	116	118	120	122	124	126	128	131	135	139	143	147	151	155	158	161	164
38	117	119	121	123	125	127	130	133	137	141	145	149	153	157	160	163	166
40	119	121	123	125	127	129	132	135	138	142	146	150	154	158	161	164	167
42	120	122	124	126	128	130	133	136	139	143	147	151	155	159	162	166	169
44	122	124	126	128	130	132	135	138	141	145	149	153	157	161	164	168	171
46	123	125	127	129	131	133	136	139	142	146	150	154	158	162	165	169	172
48	124	126	128	130	132	134	137	140	143	147	152	156	160	164	167	171	174
50	125	127	129	131	133	135	138	141	144	148	152	156	161	165	169	173	176
52	125	127	129	131	133	135	138	141	144	148	152	157	162	166	170	174	177
54	125	127	129	131	133	135	138	141	144	148	153	158	163	167	171	174	177

Average Weight of Men in Pounds with Clothes

	Feet and inches with shoes																
Age	5–0	5–1	5–2	5–3	5–4	5–5	5–6	5–7	5–8	5–9	5–10	5–11	6–0	6–1	6–2	6–3	6–4	6–5
16	109	111	114	117	120	124	128	132	136	140	144	149	154	159	164	169	174	179
18	113	115	118	121	124	128	132	136	140	144	148	153	158	163	168	173	178	183
20	117	119	122	125	128	132	136	140	144	148	152	156	161	166	171	176	181	186
22	119	121	124	127	131	135	139	142	146	150	154	158	163	168	173	178	183	188
24	121	123	126	129	133	137	141	144	148	152	156	160	165	171	177	182	187	192
26	123	125	127	130	134	138	142	146	150	154	158	163	168	174	180	186	191	196
28	125	127	129	132	135	139	143	147	151	155	159	164	170	176	182	188	193	198
30	126	128	130	133	136	140	144	148	152	156	161	166	172	178	184	190	196	201
32	127	129	131	134	137	141	145	149	154	158	163	168	174	180	186	192	198	203
34	128	130	132	135	138	142	146	150	155	160	165	170	176	182	188	194	200	206
36	129	131	133	136	139	143	147	151	156	161	166	171	177	183	190	196	202	208
38	130	132	134	137	140	144	148	152	157	162	167	173	179	185	192	198	204	210
40	131	133	135	138	141	145	149	153	158	163	168	174	180	186	193	200	206	212
42	132	134	136	139	142	146	150	154	159	164	169	175	181	187	194	201	208	214
44	133	135	137	140	143	147	151	155	160	165	170	176	182	188	195	202	209	215
46	134	136	138	141	144	148	152	156	161	166	171	177	183	189	196	203	210	216
48	134	136	138	141	144	148	152	156	161	166	171	177	183	190	197	204	211	217
50	134	136	138	141	144	148	152	156	161	166	171	177	183	190	197	204	211	217
52	135	137	139	142	145	149	153	157	162	167	172	178	184	191	198	205	212	218
54	135	137	139	142	145	149	153	158	163	168	173	178	184	191	198	205	212	219

sive. Undoubtedly many cases are being unnecessarily sent to the hospital at present because family doctors are unwilling to give their patients the simple education which so many hospitals offer, and undoubtedly many hospitals are observing diabetic patients for a longer time than is strictly necessary for the best therapeutic results.

SIMPLIFIED METHODS FOR CALCULATING DIABETIC DIETS

An intelligent patient can learn very easily how to calculate diabetic diets, how to weigh and measure food, how to test the urine for sugar and how to administer insulin.

One of Dr. Joslin's most practical contributions to the treatment of diabetes is his diet card. At the Peter Bent Brigham Hospital we have used his idea but have constructed another calculated diet card. By placing the two cards side by side one has a choice of thirty-four different diabetic diets from which it is possible to figure out in a few minutes almost any sort of food combination.

Both cards use only the simplest kinds of foods which are most easily obtainable. Dr. Joslin's card gives food mixtures with high carbohydrate low fat proportions while the Brigham card offers diets with lower carbohydrate and higher fat fractions. The Joslin card has the advantage of allowing the rapid and fairly accurate calculation of a wide range of diets.

For example; if one wishes to calculate a diet from the Joslin card containing approximately 1950 calories in 101 gms. of carbohydrate, 80 gms. of protein and 135 grams of fat one uses the carbohydrate of T. D. 2 which contains 300 gms. of 5% vegetables, 300 gms. of orange, 1 shredded wheat biscuit, 2 uneeda crackers and 120 gms. of potato, and the protein and fat from P F 11 which consists of 2 eggs, 240 c.c. of 20% cream, 30 gms. of bacon, 45 gms. of butter and 120 gms. of meat.

Or if one wishes to calculate a diet for a severe case of approximately 1000 calories containing 14 gms. of carbohydrate, 44 gms. of protein and 83 gms. of fat, one uses the carbohydrate from diet C 1 which contains 300 gms. of 5% vegetables, and the protein and fat from diet P F 6 which consists of 2 eggs, 120 c.c. of 20% cream, 30 gms. of bacon, 30 gms. of butter and 30 gms. of meat.

In order to simplify the process of education patients may be equipped with the accompanying diet lists. They should not be allowed substitutions until they have demonstrated their ability to remain sugar free on a prescribed diet at home. Then they may be taught more complicated diets if it seems desirable.

Patients may also be allowed to use the accompanying recipes. Some patients, though not all, like bran cakes, most like agar jelly, and nearly all enjoy mineral oil mayonnaise.

TABLE 2

THE HEIGHT, WEIGHT, AGE AND SEX DATA IN TWENTY-FOUR AMBULATORY CASES OF DIABETES

No.	Sex	Age	Height	Present weight	Maximum weight	Standard normal weight	Duration of diabetic symptoms	Insulin advised when first seen
1	M	64	6'1"	188	250	200	11 years	No
2	M	63	5'9"	164	201	170	4 years	No
3	F	61	5'6"	141	162	155	1 month	Yes
4	F	60	5'4"	189	275	150	5 years	No
5	F	60	5'9"	183	200	170	6 months	No
6	M	57	5'10"	215	230	175	Not known	No
7	F	55	5'3"	205	235	141	18 months	No
8	M	55	5'8"	155	180	163	Not known	Yes
9	F	55	5'4"	88	143	144	2 years	Yes
10	M	54	5'4"	134	144	145	14 years	Yes
11	M	52	5'11"	200	200	178	Not known	No
12	M	48	5'4"	148	160	144	Not known	No
13	F	47	5'6"	187	209	151	5 months	No
14	F	43	5'2"	120	137	134	17 years	No
15	M	42	5'4"	127	135	142	3 years	No
16	M	41	6'0"	161	198	180	3 years	Yes
17	F	41	5'4"	204	204	138	4 years	No
18	F	40	5'6"	171	188	146	10 months	No
19	F	36	5'5"	138	174	139	2 years	No
20	M	33	5'9"	127	127	159	3 years	Yes
21	M	32	5'5"	114	125	141	3 years	Yes
22	F	30	5'3"	163	171	127	5 months	No
23	F	16	4'10"	86	86	106	2 years	Yes
24	M	13	5'6"	97	118	120	6 weeks	Yes

THE IDEAL DIABETIC DIET

At present the ideal diet for diabetic patients is uncertain. There are certain general principles which apply to all cases, however. Weak

undernourished diabetics need to gain weight and strength. This is accomplished by high caloric feedings. Most undernourished patients will gain sufficient weight and strength if they receive 40 calories per kilogram of body weight, gram of body weight, while elderly diabetics with arteriosclerosis hypertension and chronic myocarditis do not need more than forty or fifty grams of protein a day regardless of their body weight. All diabetics like carbohydrate better

TABLE 3

DR. JOSLIN'S DIABETIC DIET CARD

Form J-13 Thomas Groom & Co., Inc., 105 State Street, Boston, Mass.

INSULIN DIABETIC DIETS

		Total Diet				Carbohydrate (C)						Protein and Fat (PF)					
	Diets	Carbohydrate	Protein	Fat	Calories	5% Vegetables	Orange	Oatmeal	Shredded Wh't	Unneeda	Potato	Egg	Cream 20% fat	Bacon	Butter	Meat	
TEST	T.D.1	181	46	44	1304	300	300	.....	3	4	240	3	120	.....	.....	.....	1
	T.D.2	101	35	43	931	300	300	.....	1	2	120	3	120	.....	.....	.....	2
	T.D.3	66	24	37	693	300	300	.....	½	2	.....	2	120	.....	.....	.....	3
	T.D.4	34	15	30	466	300	200	.....	...	...	.....	1	120	.....	.....	.....	4
MAINTENANCE	C1+PF1	14	15	30	386	300	.....	.....	...	...	.....	1	120	.....	.....	.....	1
	C2+PF2	22	19	37	497	300	100	.....	...	...	.....	2	60	.....	15	.....	2
	C3+PF3	32	24	37	557	600	100	.....	...	...	.....	2	60	.....	15	.....	3
	C4+PF4	42	29	52	752	600	200	.....	...	...	.....	2	60	30	15	.....	4
	C5+PF5	52	32	66	930	600	200	15	...	...	.....	2	60	30	30	.....	5
	C6+PF6	64	44	83	1179	600	200	30	...	...	.....	2	120	30	30	30	6
	C7+PF7	74	52	88	1296	600	300	30	...	...	.....	2	120	30	30	60	7
	C8+PF8	84	61	94	1426	600	300	30	...	2	.....	2	120	30	30	90	8
	C9+PF9	98	65	106	1606	600	300	30	½	2	.....	2	180	30	30	90	9
	C10+PF10	109	66	119	1771	600	300	30	1	2	.....	2	180	30	45	90	10
	C11+PF11	135	80	135	2075	600	300	30	1	2	120	2	240	30	45	120	11
	C12+PF12	159	84	135	2187	600	300	30	1	2	240	2	240	30	45	120	12

PETER BENT BRIGHAM HOSPITAL

DIABETIC DIET

	Grams of					Diet in Actual Food									
	Carbohydrate	Protein	Fat	Total Calories	Total Glucose	3% Vegetables	10% Fruit	Shredded Wh't	Milk	20% Cream	40% Cream	Butter	Bacon	Meat	Egg
T.D.1	148	49	43	1175	181	600	330	3	510				40		1
T.D.2	102	34	44	940	126	600	300	2		180				45	
T.D.3	67	34	66	998	93	300	300	1		120			60		2
T.D.4	34	36	79	991	63	300	200				120		30	60	1
T.D.5	15	35	90	1010	44	300					135		45	45	1
M.D.1A	10	11	6	138	17	300									1
M.D.1	14	15	38	458	27	300				120		10			1
M.D.2	22	21	53	649	39	300	80			120		20			2
M.D.3	22	31	91	1031	49	300	80			120		30	60		2
M.D.4	27	39	84	1020	58	300	130			120		15	60	30	2
M.D.5	34	47	89	1125	70	300	200			120		15	60	60	2
M.D.6	39	51	104	1296	79	450	200			120		40	45	75	2
M.D.7	48	52	118	1462	90	600	240			120		50	60	60	2
M.D.8	53	62	129	1621	102	600	200	⅓		150		50	60	90	2
M.D.9	58	60	144	1768	107	600	200	⅓		195		60	60	75	2
M.D.10	65	60	164	1976	116	600	270	⅓			180	45	60	75	2
M.D.11	76	69	182	2218	134	600	270	1			180	60	60	105	2
M.D.12	84	69	199	2403	144	600	210	1½			240	55	60	90	2

Form 194X

using for the body weight figure that obtained when insulin is started. Obese diabetics need to lose weight. This is accomplished by low caloric feeding. A stout patient does not require more than 15-20 calories per kilogram of body weight and on such a diet will lose weight surprisingly slowly. Young diabetics require a gram or a gram and a half of protein per kilo- than any other food-stuff and would rather eat less calories and more carbohydrate (in other words a high carbohydrate low fat mixture) than many calories and little carbohydrate (in other words a low carbohydrate-high fat mixture).

If one takes the trouble to obtain the height-age-weight data in a given case, one can hazard a fairly accurate guess as to the type of diet

that the patient will eventually get along well with comfortably, for by the state of nutrition one determines the calories and from the age the amount of protein necessary. The balance between the varying components of the diet can be worked out at leisure. From the point of view of routine work it is safer to prescribe a low protein, high carbohydrate-low fat mixture at first than one containing a considerable amount of fat, and it is better to underfeed than overfeed a new case.

Having planned a diet for a given case, the patient must learn how to weigh and measure it. Mild cases not needing insulin get along well with rough household measures. Any case taking insulin, however, must weigh his food accurately on food scales. Patients can learn to do this, and how to test their urines by Benedict's method in a few minutes. Thus by a short period of concentrated study it is possible to calculate and tabulate a reasonable diet for a given case on the first examination, to teach the patient how to measure and prepare it, and how to test the urine for sugar.

TABLE 4

DR. JOSLIN'S DIABETIC DIET CARD (REVERSE)

Water, clear broths, coffee, tea, cocoa shells and cracked cocoa can be taken without allowance for food content.

FOODS ARRANGED APPROXIMATELY ACCORDING TO CONTENT OF CARBOHYDRATES

	5%	10%	15%	20%
	*Reckon average carbohydrate in 5% veg. as 3%—of 10% veg. as 6%			
VEGETABLES (fresh or canned)	1%-3%	3%-5%	10% *	15%	20%
	Lettuce	Tomatoes	String Beans	Green Peas	Potatoes
	Cucumbers	Brussels Sprouts	Pumpkin	Artichokes	Shell Beans
	Spinach	Water Cress	Turnip	Parsnips	Baked Beans
	Asparagus	Sea Kale	Kohl-Rabi	Canned Lima Beans	Green Corn
	Rhubarb	Okra	Squash		Boiled Rice
	Endive	Cauliflower	Beets		Boiled Macaroni
	Marrow	Egg Plant	Carrots		
	Sorrel	Cabbage	Onions		
	Sauerkraut	Radishes	Green Peas, canned		
	Beet Greens	Leeks			
	Dandelions	String Beans, canned			
	Swiss Chard	Broccoli			
	Celery	Artichokes			
	Mushrooms				
FRUITS			Strawberries	Raspberries	Plums
			Lemons	Currants	Bananas
			Cranberries	Apricots	Prunes
			Peaches	Pears	
	Ripe Olives (20% fat)		Pineapple	Apples	
	Grapefruit		Blackberries	Blueberries	
			Oranges	Cherries	

1 gram protein,	4 calories	1 kilogram = 2.2 pounds	
1 " carbohydrate,	4 "	30 grams g. or cubic centimeters c.c. = 1 ounce	
1 " fat,	9 "	A patient "at rest" requires 25 calories per kilogram	
6.25 " protein contain	1 g. nitrogen		

30 grams 1 oz.	Carbohydrates	Protein	Fat	Calories
Contain approximately	G.	G.	G.	
Vegetables 5%	1	0.5	0	6
Vegetables 10%	2	0.5	0	10
Shredded Wheat	23	3	0	104
Uneedas, two	10	1	1	53
Potato	6	1	0	28
Bread	18	3	0	84
Oatmeal, dry wgt.	20	5	2	118
Oysters, six	4	6	1	49
Milk	1.5	1	1	19
Meat (cooked, lean)	0	8	5	77

	Carbohydrates	Protein	Fat	Calories
Contain approximately	G.	G.	G.	
Fish	0	6	0	24
Chicken (cooked, lean)	0	8	3	59
Egg (one)	0	6	6	78
Cheese	0	8	11	131
Bacon	0	5	15	155
Cream, 20%	1	1	6	62
Cream, 40%	1	1	12	116
Butter	0	0	25	225
Oil	0	0	30	270

Thomas Groom & Co., Inc., 105 State Street, Boston.

A METHOD FOR SHORTENING THE HOSPITAL TREATMENT OF PATIENTS WITH DIABETES

The necessary period of hospitalization for diabetics can be materially shortened by a preliminary course in education before entering the hospital as nowadays the urine can be made sugar free in a few hours by insulin. Insulin

TABLE 5

SOME SIMPLE DIABETIC RECIPES

BRAN WAFERS

(Use level measurements and a ½-pint tin measuring cup.)

Bran, dry-washed	3½ cups
Indfa Gum	3 level tablespoons
Saccharin	½ grain
Boiling water or coffee to make a stiff paste	
Salt	
Nutmeg or caraway seed	(As desired)

1. Mix dry ingredients thoroughly.
2. Add enough boiling water to make a mixture stiff enough to spread easily. Be sure to stir well before spreading.
3. Spread out on flat tins greased *slightly* with mineral oil. Have mixture about ⅛ inch thick.
4. Bake in a very slow oven until dry throughout.

To Prepare Washed Bran

To Wash Bran—Place bran loosely in a muslin bag and tie bag tightly to cold water faucet so that no bran will be pushed out. Turn water on and let it run. Occasionally squeeze bag, thus mixing bran and getting it wet throughout.

This process should be kept up until water coming from bran is absolutely clear.

To Dry—Spread out thinly on a tray or in pans and place in a very slow oven or on back of stove to dry thoroughly. Do not allow bran to scorch while drying.

As bran dries slowly it should be washed at least one day before it is to be used.

AGAR JELLY

1	Water	1 quart
	Agar	1 level tablespoon
2	Color (to color faintly)	
	Vinegar	2 teaspoons
	Flavoring	To taste
	(Use bottled extracts only, no fruit juices)	
3	Saccharin	1 grain

1. Mix agar-agar and water.
2. Boil 15 minutes.
3. Strain through cheese cloth to remove scum.
4. Add vinegar, coloring, flavoring, and mix.
5. Lastly, add saccharin and mix until dissolved.
6. Pour into bowls or pans and allow to harden.

MINERAL OIL MAYONNAISE

Egg yolk	1
Mineral oil	1 pint
Vinegar	2 tablespoons
Salt	½ teaspoon
Mustard	¼ teaspoon

1. Beat egg yolk until stiff.
2. Beat in 1 teaspoon of vinegar.
3. Add oil drop by drop, beating constantly.
4. Add vinegar to thin to desired consistency.
5. Add seasoning.

As much as two tablespoons may be used per meal without counting food value.

THRICE COOKED VEGETABLES

1. Prepare vegetables and cut in small pieces.
2. Put into kettle with large amount of COLD water.
3. Boil for 1 hour, then pour off hot water, draining all of it.
4. Cover vegetables with cold water and boil for ½ hour.
5. Remove hot water, again draining carefully.
6. Cover again with cold water and boil for ½ hour.
7. Drain.

Spinach, asparagus, cabbage, celery, lettuce, rhubarb, and Swiss chard so cooked may be taken without allowing for food content.

Form 196

has the specific pharmacological action of increasing the rate of oxidation of glucose in the diabetic organism. As a result, after insulin is injected the blood sugar concentration falls, glycosuria diminishes, and acidosis due to the presence of acetone bodies in the tissues disappears. The effect of insulin upon the blood sugar concentration is almost immediate; it may fall appreciably within fifteen minutes of a single small injection. Ordinarily, however, the greatest effect of a dose is not reached for about six hours. One unit is equivalent to approximately 2 gms. of excreted glucose: if a patient, for example, excretes 25 gms. of sugar in twenty-four hours on a constant measured diet, the urine will not remain sugar free on the same diet with less than twelve units a day. Patients receiving one dose a day usually take it just before breakfast with the expectation that the effect of the single dose will last until after supper, and that the night fast will prevent any marked morning hyperglycemia. Patients receiving two doses a day receive one before an early breakfast and another before a late supper so that the injections are given at approximately twelve hour intervals. Patients receiving three doses a day receive one an hour before breakfast, another just after dinner and the third dose at nine o'clock at night with the idea of so spacing the insulin that the blood sugar curve is kept as even as possible during the entire twenty-four hours. This method, proposed by Allen, appears to allow severe cases more food and less insulin than any other method of timing injections and does not cause reactions.

I am convinced that many cases are being sent to the wards of our various hospitals for insulin which could be treated just as efficiently and far more economically as out patients. I have made a few clinical experiments in an intensive method of treatment and education for this type of case. The following is an example of what can be accomplished in a short space of time.

A man 41 years old, six feet tall, weighing 161 pounds, came to the hospital six months ago. His diabetes was of three years' duration during which time he had lost thirty pounds in weight and had almost constantly excreted sugar despite a conscientious attempt at diet. Since he was definitely underweight and not strong enough to be economically efficient he seemed to need insulin. His physical examination was negative. A specimen of urine contained 2% of sugar and the blood sugar concentration was .19%. He was taught at the first visit how to measure and prepare an analyzed diet and was

sent home for three days with a diet containing 76 gms. of carbohydrate, 69 gms. of protein, 182 gms. of fat, yielding 2200 calories. He was given this diet to allow one gram of protein per kilogram of body weight, a little over 25 calories per kilogram of body weight, and a fairly palatable amount of carbohydrate. He was told not to test his urine for sugar but to bring to the hospital four days later after dinner a complete 24 hour specimen of urine.

When he returned to the hospital for his second visit the twenty-four hour urine was found to contain 12 gms. of sugar, and a single specimen 2.5%. As the urine was not sugar free, the patient was given 10 units of insulin. The effect of the drug on the blood and urine is shown by the accompanying table:

TABLE 6

THE EFFECT OF 10 UNITS OF INSULIN UPON BLOOD AND URINE SUGAR

Time P. M.	% of sugar in urine	Blood sugar concentration	
12:35	2.5	.18	10 units of insulin
1:35	1.4		
2:35	Trace		
3:20	0	.10	

After the urine became sugar free the possible danger of an insulin reaction was stopped by an orangeade containing 20 gms. of sugar. While he was becoming sugar free, the patient was taught how to measure and inject insulin and was made to read and pass an examination upon the signs, symptoms and treatment of insulin shock, and upon the care of the common complications of diabetes. At the end of the afternoon he was sent home with instructions to continue with the same diet but to take 8 units of insulin each morning when he got up.

A week later he reported for his third visit with a twenty-four hour sample of urine. This contained a trace of sugar. The blood sugar concentration was now .15%. The patient was told to take 10 units of insulin a day instead of 8 and to report in another week for a fourth visit. The urine was now sugar free and the blood sugar concentration was .14%. Thus four office visits in this instance were sufficient to render the urine permanently sugar free, to establish a diet compatible with efficient living, to instruct the patient how to prepare and measure this diet, how to test the urine, how to take insulin, and how to take care of himself.* In other words, as striking a therapeutic result was accomplished without loss of time to the patient as is usually accomplished in two or three weeks of hospitalization.

*The patient reports that he has been sugar free practically all the time since his last visit to the hospital, that he is feeling well, and "working like the devil" on 10 units of insulin a day.

I believe that some such plan of intensive treatment could be safely perfected in the diabetic clinics of some of the various out-patient departments of our hospitals and is feasible in the office work of any one familiar with the methods of diabetic management. Much time could be saved for many diabetic patients in this way and the hospital beds available for diabetics could be used for needier and sicker patients where bed care is of particular importance.

DIABETIC COMA

Coma is the chief diabetic emergency which the general practitioner is called upon to treat. During the last year I have seen seven cases which emphasize certain practical points in regard to the diagnosis, prognosis and treatment of this condition.

Of the seven cases, four occurred in young people less than twenty years old. This fact shows that diabetic coma complicates juvenile diabetes more frequently than the diabetes of elderly people. On the other hand, no diabetic is immune.

Of the four juvenile cases, diabetes was unsuspected in three until the patients became unconscious. This fact shows that the onset of diabetes may be insidious and that coma may develop very acutely so that the diagnosis is not always easy.

Two cases developed coma from carelessness. This fact shows the importance and difficulties of education in preventing coma. One of these cases was a boy whose elder sister died of diabetes. His mother knew the dangers of diabetes and yet considered herself unable to make her son follow out any prescribed course of dieting. The other case came to the hospital in coma six months ago because she decided not to follow a diabetic diet. She recovered and reëntered in coma a second time a few months later because she ran out of insulin and thought she could get along without it!

Two cases developed coma as a result of what seemed to be slight infections. This fact shows that even the mildest infection is of importance to the diabetic and must be taken seriously. It is difficult to make diabetic patients realize that a head cold or a sore throat or a corn may rapidly develop into a fatal illness.

THE PHYSICAL SIGNS OF DIABETIC COMA

There are certain physical signs which are important in the diagnosis of coma. The temperature is apt to be subnormal, the pulse weak, rapid and of low tension. The skin is dessicated and there is usually a characteristic redness to the lips and cheeks. The eyeballs are soft. The respiration may be slow or slightly elevated but is abnormal in depth varying from slight hyperpnoea to outspoken air-hunger. The knee jerks may be absent although there is not a rigid neck

or Kernig's sign. A well trained nose can often detect the fruity acetone breath. The urine contains sugar. Albumen and showers of casts are frequently found. Acetone and diacetic acid may both be absent although both acids are usually present in large amounts.

THE PROGNOSIS OF DIABETIC COMA

The prognosis of coma is always uncertain. Three of this series of cases died while four recovered. Of the fatal cases, two were comatose for twenty-four hours, and one for fifteen hours before receiving insulin. Of the four recovering cases, none were comatose for more than twelve hours before treatment was started. This fact shows the importance of early diagnosis and treatment. In the absence of an overwhelming infection such as a general septicemia most coma cases will recover with insulin. In the presence of severe infection insulin is practically inert. The longer the state of coma before insulin is given, the greater the chance for a terminal infection to develop.

In two fatal cases, blood pressure readings could not be obtained and in the third the systolic pressure was 80 mm. with a diastolic pressure of 40 mm. All four recovering cases had systolic pressures greater than 90 mm. with diastolic pressure greater than 50 mm. This observation suggests that the lower the blood pressure is when the case is first seen the worse is the prognosis.

In the three fatal cases, the blood sugar readings were greater than .80% while in the four recovering cases the blood sugar readings were not above .50%. Apparently, cases with very marked hyperglycemia do not respond to insulin as readily as do these with more nearly normal figures.

Finally, the degree of acidosis as measured by the alkali reserve of the plasma was not of particular prognostic significance. Of three cases in which the determination was made, the one with the lowest alkali reserve recovered.

On the whole, the diagnosis of diabetic coma must often depend upon the physical examination rather than upon the history. All cases have sugar in the urine and all have acidosis. Coma, in the presence of glycosuria but in the absence of acidosis, is probably not diabetic coma. Acidosis can be recognized to a certain extent by the clinical appearance of the patient, more definitely by the type of breathing, but positively by a determination of the carbon dioxide tension of the alveolar air or by the alkali reserve of the blood plasma. The prognosis depends to a large extent upon the duration of the symptoms, so that the earlier the diagnosis is established and treatment is instituted, the better is the outlook. Patients in shock with a very low blood pressure and marked hyperglycemia have a poorer outlook than do those with a well sustained blood pressure and lower blood sugar concentrations; the factor of infection is of great importance.

THE TREATMENT OF DIABETIC COMA

The treatment of diabetic coma is simple. In the Peter Bent Brigham Hospital the following routine is used, modified in minor details for each case. As soon as possible after admission to the hospital the patient is placed in a warm bed and is surrounded by hot water bottles or electric pads in order to bring the body temperature to normal. A specimen of urine is obtained by catheter if necessary and the lower bowel is washed out by an enema. Fluids are immediately given under the skin and by rectum, 500 or 1000 c.c. of normal saline being given as a subpectoral and tap water, 5% glucose, 5% glucose with 5% sodium bicarbonate or sodium bicarbonate solution alone being given by the Murphy Drip method. A dose of 25-50 units of insulin is given subcutaneously and is repeated at hourly intervals as long as is necessary.

The effect of insulin is observed by repeated urine tests. As the percentage of sugar in the urine falls, the amount of insulin given is diminished. When the patient becomes conscous he is allowed orange juice or oatmeal gruel by mouth, a diet which is gradually increased until finally a low diabetic diet is given. We no longer use gastric lavage as a routine, although if the patent is nauseated and vomits the stomach is washed. For the first few days, very small amounts of food and repeated insulin injections are given which are controlled by frequent urinalyses to be certain that the urine is constantly sugar free. As the patients regain tolerance the insulin is diminished and the food is increased, until finally one of the standard diabetic diets is given with whatever insulin is necessary.

The effect of insulin in patients who recover is almost immediate. The temperature rises, the pulse and respiration rates fall and there is a remarkable subjective improvement. For a few days the temperature remains above normal and the pulse rate is elevated, suggesting that even in recovering cases there may be an underlying acute infection. Then the temperature and pulse become normal and the patient makes an uneventful convalescence. There is nothing more gratifying than the successful treatment of coma with insulin.

SUMMARY AND CONCLUSIONS

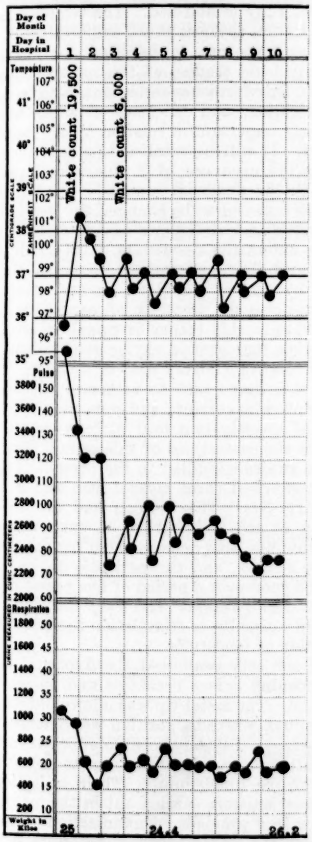
Insulin is a well established aid in the treatment of certain cases of diabetes. Since the majority of diabetic patients in this country depend upon their family doctor for treatment, general practitioners must know how and when to use insulin.

Diabetes tends to be a mild disease and only about one-third of the cases ordinarily seen need

TABLE 7

CLINICAL CHART OF A RECOVERING CASE OF DIABETIC COMA

A girl 13 years old—Coma of 12 hours' duration before treatment



insulin. Undernourished diabetics, young diabetics, diabetics with a surgical complication and diabetics with acidosis or an acute infetion usually need insulin, while elderly and obese diabetics do not.

Methods have been developed for simplifying the treatment of diabetes. At present there is a tendency for too many diabetics to be sent to hospitals for education and treatment. Patients can now learn readily how to calculate diets, how to measure and prepare them, how to test the urine for sugar and how to administer insulin. Insulin affords a rapid method for desugarization. Selected cases can be intensively treated with insulin, so that a satisfactory therapeutic result can be accomplished in a few hours instead of in a few days or weeks. Attempts should be made to shorten the period of hospital treatment for diabetics because of the expense of treatment in the first place and so that the available hospital beds for diabetics can be used by sick and needy patients who require bed and special nursing care.

Coma is the chief diabetic emergency which the general practitioner is called upon to treat. Coma tends to occur in young people more frequently than in old people, it may begin acutely without known antecedent symptoms of diabetes, it can usually be prevented by properly trained diabetics. The physical signs of coma are important in the early diagnosis and there must be manifestations of acidosis and glycosuria to justify the diagnosis. Coma must be treated as soon as possible after the diagnosis is established for the longer the duration of coma before treatment is instituted the worse the prognosis. Coma cases with infection, those in shock, and those with very high blood sugar concentrations have a poorer outlook than those without demonstrable infection with a well sustained blood pressure and without tremendous hyperglycemia. The treatment of coma is simple and satisfactory. Large doses of insulin, plenty of fluid and good nursing care are the essentials.

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STUDIES ON THE MECHANISM OF EXTERNAL PANCREATIC SECRETION

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THE present communication reports the results obtained from a study of the chemical reaction, as measured by the hydrogen ion concentration, of the first portion of the duodenum.

It has been commonly assumed by physiologists that gastric chyme, after entering the duodenum, maintains a relatively high degree of acidity until churned up with alkaline duodenal contents. On this premise physiologists based important theories, among which are the well known "acid control of the pylorus" and the "secretin" theories. The two theories men-

tioned became generally accepted by physiologists as correct. The clinician, always interested in important physiological discoveries, consequently accepted these theories and attempted to apply them as bases for explaining phenomena observed in pathologic subjects. This state of affairs existed in clinical medicine until Cole[1] first questioned the application of the theory of the "acid control of the pylorus" to man. Later McClure, Reynolds and Schwartz[2] produced strong evidence that acidity did not govern the action of the pyloric sphincter in man. The observations of these investigators on man and those of Carlson[3] and coworkers on animals have thrown serious doubt on the correctness of the theory of the "acid control of the pylorus." In order to explain phenomena occurring in that medically highly important region, the pylorus, it is essential to establish fully whether or not the pyloric sphincter is governed by the acidity of that region. Data obtained in the present investigation bear on this question.

The development of the fluoroscope and the duodenal tube make it possible to obtain hepatic and pancreatic secretions. These secretions are, therefore, available for both physiological and pathological studies. This allowed McClure, Wetmore and Reynolds[4] to devise methods for ascertaining the external enzymic function of the pancreas, and to obtain data of both physiologic[5] and pathologic[6] interest. The physiological data[5] obtained showed that the secretion of pancreatic enzymes is stimulated more powerfully by foodstuffs than by water; and that the degree of this stimulation is not demonstrably affected by the chemical reaction, the hydrogen ion concentration, of the duodenal contents. McClure, Montague and Campbell[7] established procedures for applying the potentiometer to the determination of the hydrogen ion concentration of duodenal contents. Their observations, together with those of McClure, Mortimer and Montague[8], show that the pancreas in achylia gastrica reacts normally to the stimulation of foodstuffs. These observations throw doubt on the application of the "secretin" theory and consequently reopen the question as to what factor or factors actually stimulate the secretion of pancreatic juice.

The hepatic fraction of duodenal contents has received much attention from clinicians since the first publication of Lyon[9] on the subject. The present status of this work is fully discussed in a recent publication by Jones[10]. McClure and coworkers[11] have established procedures for ascertaining the state of liver function from the examination of the biliary fraction of duodenal contents. While the work of all these investigators yield much of clinical interest, it is open to a serious criticism. This criticism is that magnesium sulphate solution, employed as a stimulant to the flow of bile, does not always cause such stimulation in the normal person[11]. It is, therefore, important to find some substance which will uniformly stimulate the flow of bile in normal persons.

From the above discussion it is obvious that the factor, or factors, which normally stimulate the secretion of the pancreatic juice and of bile are not fully established. The establishment of such factors assumes much importance if duodenal contents are to be used for physiologic or pathologic studies of the pancreas and the liver. This furnishes the reason for the present investigation, and the data obtained are particularly important in relation to the "secretin" theory. It will be recalled that this theory assumes the existence of a substance "prosecretin" in the duodenal mucosa. The "prosecretin" is assumed to be changed to "secretin" by the action of hydrochloric acid of gastric chyme, which enters the duodenum. "Secretin," thus developed, is then assumed to reach the pancreas by way of the blood stream, after which it stimulates the secretion of pancreatic juice.

Obviously this theory is premised on the assumption that gastric chyme renders the duodenum relatively highly acid in reaction. Therefore, it is important to ascertain whether this assumption is correct; and data obtained in the present investigation bear directly on the question of the correctness of this assumption.

The subjects studied in the present investigation comprised three normal men and a woman of thirty-five. This woman had the condition known as achylia gastrica, of unknown origin. No cause has been demonstrated for the presence of the achylia, and the patient's health has remained good over a period of five years' observation. The pancreas[8] and liver[12] of this patient reacted normally to stimulation produced by the ingestion of foodstuffs.

All subjects swallowed the duodenal tube in the morning before breakfast and on an empty stomach. The metal tip was allowed to enter the duodenum, where its position was determined by fluoroscopy. Duodenal contents for enzymic concentrations were obtained from the second portion of the duodenum. Their collection and examination were carried out in the manner described in a previous communication[5]. Duodenal contents for pH determinations were obtained by aspiration from the first portion of the duodenum; the metal tip was either just beyond the distal end of the sphincter, or in the sphincter itself. The duodenal specimens for pH estimations were collected over periods of a few seconds to several minutes, depending on the time necessary to collect a quantity sufficient for potentiometric purposes: about 5 c.c. The preservation of the specimens and the technic of the pH measurements were described in a previous communication[7]. At the conclusion of an experiment the subject was given a barium meal. This permitted verification of the location of the metal tip of the tube. Without the use of

the barium meal, experience permits the exact location of the metal tip of the tube. The senior author was able to prove this to an experienced but most skeptical radiographer and to his unqualified satisfaction.

For purposes of the present investigation the horizontal fluoroscopic table was found to be indispensable. Using this table and by proper manipulation of the tube the metal tip of the duodenal tube could be kept in one position indefinitely.

The pH of duodenal contents obtained from the duodenum immediately distal to the sphincter was determined in three normal subjects. To stimulate the flow of pancreatic juice, these subjects ingested a mixture of twenty-five c.c. each of olive oil and tap water. While titration demonstrates that olive oil produces a free flow of acid gastric juice, this phenomenon was verified by potentiometric determinations in one of the subjects (See Table 2.) The times of collections of duodenal contents and the pH of the various specimens from these subjects are outlined in the following table (Table 1).

TABLE 1

pH of Contents of Portion of Duodenum Immediately Distal to Sphincter and Obtained After Ingestion of Olive Oil

Subject	pH of duodenal contents	Period of collection	Color of duodenal contents	Time and material ingested	Position of tip of duodenal tube
B				10:03; 25 c.c. each olive oil and water	
	6.364	10:08-10	yellow		Duodenum
	6.202	:14-15	brown		"
	6.381	:20-22	"		"
	6.812	:26-28	"		"
	6.807	:31-33	"		"
	6.851	:35-37	"		"
	6.747	:42	"		"
	7.202	:50	"		"
	7.009	:53-54	"		"
	7.291	11:00-02	"		"
D				9:54; 25 c.c. each olive oil and water	
	6.252	10:02-08	yellow		Duodenum
	7.004	:12-25	"		"
	6.687	:25-38	brown		"
	6.435	:38-45	"		"
	7.261	:45-54	"		"
	6.781	:54-59	"		"
	6.531	:59 to 11:05	"		"
	6.500	:05-08	"		Sphincter
	5.400	:09-13	"		"
	6.003	:13-19	"		"
	6.254	:19-25	"		Duodenum
	6.405	:25-28	"		"
	6.388	:28-32	"		"
O				8:46; 25 c.c. each olive oil and water	
	7.207	9:25-27	brown		Duodenum
	8.556	:40-45	"		"
	8.161	:45-51	"		"
	8.406	:51-56	"		"
	8.105	10:00-06	"		"
	7.422	:06-08	"		"

TABLE 2

pH of Contents of Duodenum Immediately Distal and of Stomach Just Proximal to the Sphincter and Obtained After the Ingestion of N/10 HCl or of Olive Oil by Mouth

Subject	pH of duodenal contents	pH of gastric contents	Period of collection	Time and material ingested
O				9:17; 100 c.c. N/10 HCl
	1.799		9:21-22	
	3.834		:23-24	
	8.652		:30-32	
	8.136		:40-42	
	8.789		:47-58	
O				9:00; 100 c.c. N/10 HCl and gum arabic
		2.520	9:00	
		2.430	:15	
	3.160	1.691	:30	
	9.055	1.531	10:00	
	8.472	2.745	:30	
D				10:02; 25 c.c. each olive oil and water
	6.675		10:15-18	
	8.342		:28-35	
	7.189		:37-44	
		1.821	:44	
		1.892	:50	
	6.626		:57	
		2.695	11:02	
		2.323	:05	
		2.221	:08	

A large number of observations, some of which have been published[5], establish conclusively that in normal persons the onset of the flow of pancreatic juice coincides with that of the bile. After the ingestion of olive oil, the onset of stimulation of the bile flow is evidenced by an abrupt change in color of the duodenal contents from light yellow to dark brown. On the basis of this criterion; before stimulation of the pancreas occurred in Subjects D and B (Table 1), the pH of the duodenal contents showed them to be either slightly acid or slightly alkaline; the actual pH values ranged from 6.252 to 7.004. After stimulation of the pancreas occurred, the pH values of the duodenal contents of Subject O showed persistently alkalinity. The pH values of the contents of Subjects D and B showed

either slight acidity or alkalinity; while specimens taken from within the pyloric sphincter (Subject D) were slightly acid, but only one value differed materially from those of the duodenal specimens.

The type of experiment outlined in Table 1 was repeated, the subjects drinking solutions of hydrochloric acid instead of olive oil and water. The acid was given either as the aqueous tenth normal solution or after mixing this solution with sufficient gum arabic to form a paste. Representative experiments are outlined in the following table (Table 2). In one experiment the subject ingested olive oil and water and then observations on the pH of both the stomach and first portion of the duodenum were made.

During the acid ingestion experiments, such as outlined in Table 2, the gastric contents showed pH values ranging from 1.434 to 3.532. Over an initial period, after the ingestion of the acid, of fifteen to thirty minutes the duodenal contents showed pH values varying from 1.799 to 3.773. After this initial period the duodenal contents usually became alkaline and remained so; the pH values ranging from 8.132 to 8.799. However, in one subject the pH values ranged from 6.125 to 6.683; which shows that the initial acidity was decreased. The experiment with olive oil shows that it produces a free flow of acid gastric juice.

The enzymic concentrations of duodenal contents obtained after the ingestion of olive oil and the various acid mixtures were determined. These determinations were made in duodenal contents obtained over comparable periods of collection in the manner fully described in a previous communication[5]. Results, representative of these obtained in a series of such experiments, are outlined in the following table (Table 3).

TABLE 3

ENZYMIC CONCENTRATION AND PH OF DUODENAL CONTENTS OBTAINED AFTER THE INGESTION OF ACID AND OF OLIVE OIL

Subject	Enzymic concentration of duodenal contents: Proteolytic in mgms.	Amylolytic in mgms.	Lipolytic in c.c.	pH of duodenal contents	Substance ingested by mouth
O	5.7	2.5	2.5	8.161	25 c.c. each of olive oil and water
	2.7	1.4	0.7	—	50 c.c. N/10 HCl solution
	2.2	0.2	0.0	3.218	60 c.c. N/10 HCl and gum arabic
	4.3	1.2	1.2	8.472	100 c.c. N/10 HCl and gum arabic
D	3.6	1.6	1.2	7.016	25 c.c. each of olive oil and water
	2.5	1.4	0.5	6.125	110 c.c. N/10 HCl and gum arabic

Study of the table shows that the greatest enzymic concentrations were obtained in duodenal contents derived after the ingestion of olive oil. In one experiment on Subject O, of Table 3, the enzymic concentrations and the pH values were very low. This shows that but slight stimulation of the pancreas had been produced by the acid mixture ingested, although the presence of gum arabic in the duodenal contents proved that acid was entering the duodenum. These results confirm those obtained in a previous investigation[5]. The results of that investigation showed that olive oil was a more powerful stimulant of the pancreas than either protein or carbohydrate; and that water produced less stimulation than did foodstuffs. As is well established, water stimulates the production of acid gastric secretion. Therefore, the ultimate conditions produced by water drinking are the same as those effected by the ingestion of tenth normal hydrochloric acid solution.

In addition to the experimental work outlined above, a series of experiments were carried out on the subject with achylia gastrica. A part of these studies yielded results which showed that the pancreas reacted to stimulation the same as that of normal persons, and has been published[8] elsewhere. The unpublished studies on this subject have consisted in the feeding of foodstuffs so prepared as to be neutral, acid or alkaline in reaction. Alkalinity was produced by adding tenth or twentieth normal sodium hydrate solution to the food, and acidity by using tenth normal acid. Sufficient sodium hydrate was employed to maintain a slight degree of alkalinity of the gastric contents, as determined by potentiometric estimations; the actual pH values of the gastric contents varied from 7.093 to 8.898. The addition of either acid or alkali had no demonstrable effect on the enzymic function of the pancreas. On one occasion stimulation of the pancreas followed the ingestion of gum arabic, which had been mixed with sufficient sodium hydrate solution to keep the gastric contents alkaline. The experimental procedure governing these studies was the same as that used in those already published[8]. For this reason it is considered unnecessary to tabulate them.

Also, studies on normal persons have been carried out which show that peptone, dextrose or oleic acid, when introduced into the duodenum, will stimulate a normal flow of pancreatic juice, as judged by measuring the enzymic concentrations of duodenal contents. These substances were dissolved or suspended in water of sufficient quantity to make a total volume of 50 c.c. The amounts used were as follows: 10 grams of peptone made from beef; 25 grams of chemically pure dextrose; two to 10 c.c. of oleic acid.

Unquestionably smaller amounts of these substances would stimulate the pancreas, but no attempt was made to ascertain the minimal quantities which would do so. The small amount of oleic acid necessary to stimulate the pancreas could be manufactured from the fat and oil of the food ingested with almost any type of meal usually eaten.

DISCUSSION

The studies outlined in Table 1 show that acidity of gastric chyme is largely neutralized the instant it reaches the duodenum. The neutralization may be compared to that produced by pouring an acid solution onto the surface of an alkaline fluid. In other words, the observations show that the acidity of gastric chyme, thrown into the duodenum, is neutralized without first rendering the duodenal contents relatively highly acid; which the "secretin" theory assumes. Thus, the findings negative a most essential premise of the "secretin" theory.

The studies show that the ingestion of acid is followed by stimulation of the pancreas. This stimulation may be explained on the basis of the "secretin" theory, since duodenal contents are initially relatively highly acid in reaction after the ingestion of acid. However, in Subjects D and B of Table 1 the duodenal contents were either only faintly acid or even alkaline during the corresponding initial period after the ingestion of olive oil. Thus, if "secretin" was formed, its formation occurred during a very short period in which the duodenum was but faintly acid in reaction.

Hydrochloric acid could stimulate the pancreas through the working of a nervous mechanism, the existence of which has been established in animals. On the other hand, the stimulating effect following the ingestion of acid may have resulted from the absorption of disintegration products of mucin, cellular debris, etc., thrown into the duodenum from the stomach under the experimental conditions of these studies; which have been discussed in a previous communication[5]. The absorption of chlorides, formed after the ingestion of acid, might stimulate the pancreas; for it has been found that solutions of sodium phosphate and of magnesium sulphate will often do so, when introduced into the duodenum[12]. Thus, the stimulation of the pancreas following the ingestion of acid may be satisfactorily explained on some other basis than that of the "secretin" theory.

It is well established that under normal conditions the fasting stomach often contains a small amount of acid fluid, usually not over 50 c.c. At the beginning of a meal this fluid is rapidly diluted through mixing with the food initially ingested; the actual occurrence of this process has been demonstrated by the senior author by fluoroscopic observations[12]. The consequent dilution of the acid gastric contents greatly decreases the normality of its acidity. Furthermore, McClure, Reynolds and Schwartz[2] have shown that the food begins to leave the stomach as soon as it has been ingested. Thus food does not remain in the stomach until acidified with a copious secretion of newly formed gastric juice. The slight acidity of food initially reaching the duodenum is confirmed by the observations on Subjects D and B of Table 1. Furthermore, McClure, Montague and Campbell[7], and Hume, Denis, Silverman and Irwin[13] have found that fasting duodenal contents are either alkaline or nearly so, and the mixing of food thrown over into the duodenum with the contents already there would further reduce the normality of the gastric chyme. These findings do not support the theory that acidity is an essential factor in producing the stimulation of the flow of pancreatic juice.

That the flow of pancreatic juice does not continue indefinitely, after having once been established, is shown by the studies of McClure and Wetmore[5]. These investigators found that when small meals had been ingested pancreatic stimulation diminished after an hour or two; while after the ingestion of ordinary sized meals the flow of pancreatic juice continued unabated for hours. The contents of the duodenum during this time were either alkaline or nearly so, as shown in this and previous investigations[5], [7] and by the work of Hume, Denis, Silverman and Irwin.* Furthermore, the findings in the present investigation show that even the extreme proximal portion of the duodenum was never more than faintly acid after the stimulation of the pancreas had been established. These findings indicate that the role played by acidity in maintaining the secretion of pancreatic juice would seem to be inconsequential.

In the subject with achylia gastrica, acid, alkaline or neutral foods produced comparable stimulating effects on the pancreas. Also, this subject's pancreas reacted in an apparently normal manner under conditions which kept the gastric contents slightly alkaline, as shown by potentiometric measurements. These findings give indisputable evidence that pancreatic stimulation can occur in the absence of acidity of the stomach and duodenum; and this supports the conclusion drawn in the preceding paragraph. It will be recalled that this subject's pancreas reacted to stimulation similar to that of a normal person.

The large amount of work stimulated by the publication of Bayliss and Starling on the subject of "secretin" has fully established that the

*During the earlier studies on pancreatic function by the author and coworkers the use of pure proteins, fats and carbohydrates was advised by Dr. Lafayette B. Mendel. Whatever merit that and subsequent studies on the subject possessed has been largely due to Dr. Mendel's suggestion. For this reason regret is expressed that the highly important work of Hume, Denis, Silverman, and Irwin on the pH of the duodenum was not supplemented by the use of pure representatives of the various types of foodstuffs. In our experience a predominating type of foodstuff in a meal does not always give the same result as when that type of foodstuff is fed in a pure state.

injection of tissue extracts intravenously, and also the products of protein decomposition, can stimulate the secretion of pancreatic juice. A perusal of this work suggests that certain classes of the substances injected stimulate the pancreas to different degrees of activity; for example, von Fürth and Schwartz[14] found that cholin caused less stimulation than did an extract of intestinal mucosa. In the present investigation it has been found that peptones, dextrose and oleic acid, which represent products of food digestion, will stimulate the pancreas after placed in the duodenum. Further, in this and previous investigations[6] it has been found that pure fat stimulated a greater flow of pancreatic juice than did either pure protein or carbohydrate, and that foodstuffs stimulated a greater flow than did water or acid. These differences noted in the stimulating properties of various substances would not be expected to occur if a nervous mechanism were the principal factor which stimulated the pancreas under usual conditions. This deduction, together with the findings that intravenously injected substances and digestion products placed in the duodenum stimulate the pancreas, suggests that the absorption of products of food digestion is usually the essential factor in stimulating the secretion of pancreatic juice.

The findings show that the initial portion of the duodenum is persistently alkaline or nearly so. This observation would seem to furnish the last link in the chain of evidence which shows that acidity does not govern the action of the pyloric sphincter.

SUMMARY

The above discussion may be summarized as follows:

a. Gastric chyme is neutralized, either completely or nearly so, immediately on leaving the stomach and in such manner that the duodenum is never more than faintly acid under normal conditions of eating. This negatives a most essential premise of the "secretin" theory.

b. The onset of flow of pancreatic juice may be explained satisfactorily without employing the "secretin" theory; even after rendering the duodenum relatively highly acid by means of feeding acid.

c. The findings indicate that the role played by acidity in maintaining the secretion of pancreatic juice throughout the period of digestion is inconsequential.

d. The flow of pancreatic juice can be stimulated in an approximately normal manner in the entire absence of acidity of either the stomach or duodenum.

e. Intravenously introduced substances can stimulate the flow of pancreatic juice.

f. Products of food digestion stimulate the flow of pancreatic juice after having been placed directly in the duodenum.

g. The observations throw further doubt on the validity of the theory of the "acid control of the pylorus."

CONCLUSIONS

1. The findings indicate that the duodenum is normally alkaline or nearly so after the ingestion of food. This negatives a most essential premise of the "secretin" theory.

2. The findings indicate that the products of food digestion are the essential factor in causing the stimulation of the secretion of pancreatic juice rather than the formation of "secretin."

3. The observations furnish the last link in the chain of evidence which shows that acidity does not govern the action of the pyloric sphincter.

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AGE AS A FACTOR IN DIABETIC MORTALITY

BY J. W. THOMSON, M. D.

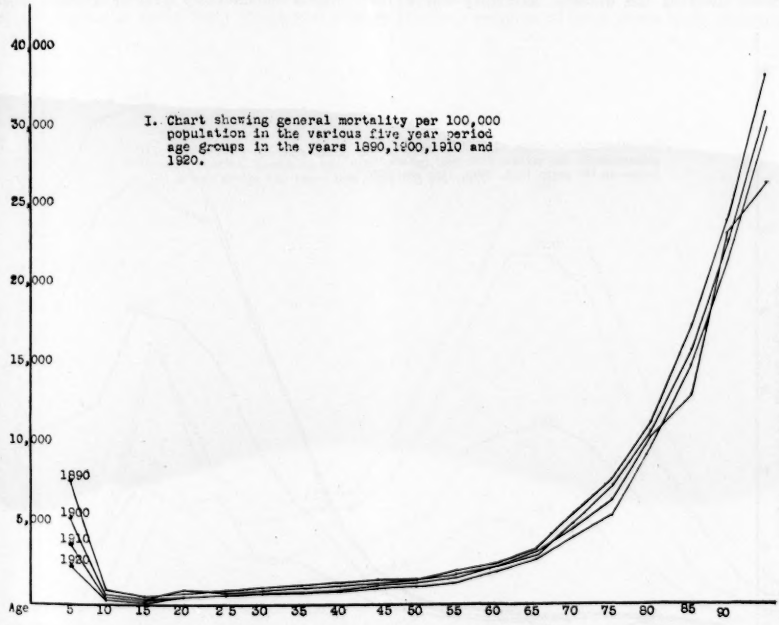
[From the Diabetic Clinic of the New England Deaconess Hospital.]

It has long been apparent that there is a definite age period at which diabetic mortality begins to rise rapidly. Figures have been published showing that this rise probably begins at about the age of forty-five years. However it has not been determined as to whether or not this diabetic mortality continued to rise progressively with age, similar to general mortality, or whether there was a definite age period when it reached its height and then fell.

This statistical study was undertaken with the idea of determining at what age period the diabetic mortality is greatest and also at what age periods diabetic mortality is greatest as com-

pared with general mortality. To determine this it was necessary to tabulate the general and diabetic mortality during one year of each of the past four decades, namely, 1890, 1900, 1910, and 1920, in order that these figures might be representative of these four decades and to eliminate the possibility of any variations due to other influences such as epidemics, etc., which may have occurred during a single year.

From these data graphs were drawn showing general mortality, diabetic mortality and diabetic as compared with general mortality rates



I. Chart showing general mortality per 100,000 population in the various five year period age groups in the years 1890,1900,1910 and 1920.

in the five year age groups given in the Mortality Statistics of the United States Census Reports during the four years considered. In attempting an interpretation of these graphs there are therefore two distinct considerations: First the curves for a decade with their changes according to the age groups and; Second the curves for each of four decades compared with one another.

GENERAL MORTALITY

In this graph the most striking feature is the general tendency during the four decades for a drop in the general mortality during the period of life from five to ten and next from ten to fifteen years of age. Following this period of lowered mortality there is a slight but steady rise up to the fifty-five to sixty year period when the curve makes a very sudden and rapid rise and continues the ascent to the period above one hundred years.

The only very evident difference in the curves for the four decades occurs at the period from birth to five years of age and here there is a decided decrease in the death rate. For example we find that in 1890 the mortality during this period of life was 7,914.72 per 100,000 population, while in 1920 during the corresponding period of life the mortality rate had dropped to 2,612.55 per 100,000.

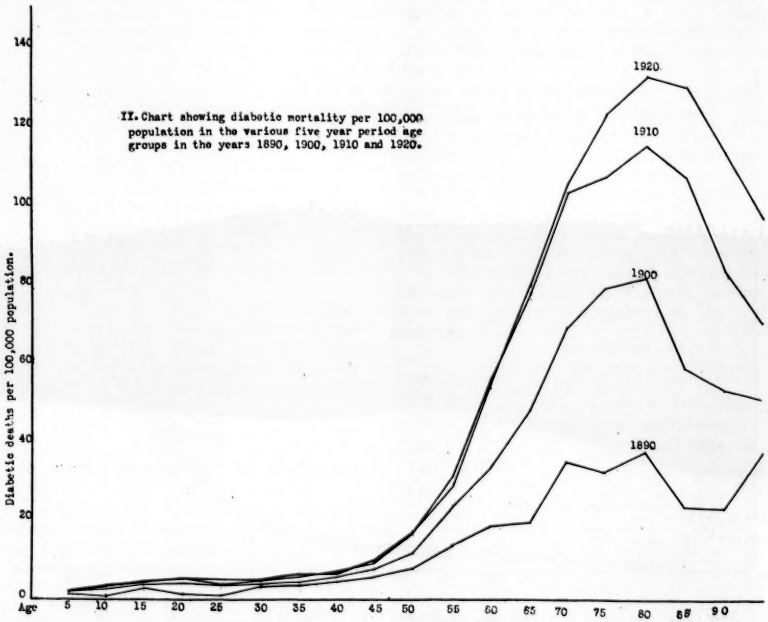
It is also evident from these curves that there is a tendency toward a general drop in mortality in all age groups from 1890 to 1920 with the curve for 1920 showing a latency in rise at the age of seventy to seventy-five. This latency in the rise of general mortality during these later age groups may however be explained by the fact that this curve does not in any way take into consideration the years of the influenzal epidemic. This drop may therefore be entirely a post-influenzal effect. That is to say, during the period of the epidemic there must have been a rise in general mortality and this rise would logically be followed by a period of decreased mortality due to the fact that the weaker individuals had previously succumbed during the epidemic.

DIABETIC MORTALITY

The diabetic mortality curves for the four decades run a comparatively parallel course up to fifty years of age, and show for all four decades only a slight but constant rise from birth up to forty-five years. Beginning at forty-five years however there is a sudden marked rise in the death rate up to seventy when the curve begins to flatten out, reaching its height at eighty years. Following this maximum at eighty there is a sudden drop in the number of diabetic deaths per population. This flattening out and eventual drop of the diabetic mortality curve are without doubt explainable by the fact that the more severe diabetics have succumbed earlier to the disease, while those cases occurring or persisting during these later periods of life are in the majority of instances probably of the more mild arteriosclerotic type in whom death is recorded to have taken place from some other cause.

One very suggestive fact to be noted in these curves is that the diabetic mortality was practically the same up to seventy years for 1910 and 1920. In 1920 the curve after seventy continues upward while that for 1910 does not rise as rapidly. In the light of the knowledge that diabetic mortality is on the increase in the United States it seems probable that this fact could be logically explained by the greater success in clinical diagnosis and treatment prolonging the life of the individual.

There is on the whole a general rise in diabetic mortality from 1890 to 1920, however there is no appreciable increase in 1920 over 1910 until after the age of seventy has been reached. Another possible explanation of this approximation of the curves of 1910 and 1920 may be found in considering this as another post-influenzal manifestation. That is to say, were we to consider similar curves for 1918 we might find that there was a considerably greater diabetic mortality during 1918 than in 1920, the more severe diabetic having succumbed to the disease during the influenzal period and resulting in a lowered mortality following this period.



II. Chart showing diabetic mortality per 100,000 population in the various five year period age groups in the years 1890, 1900, 1910 and 1920.

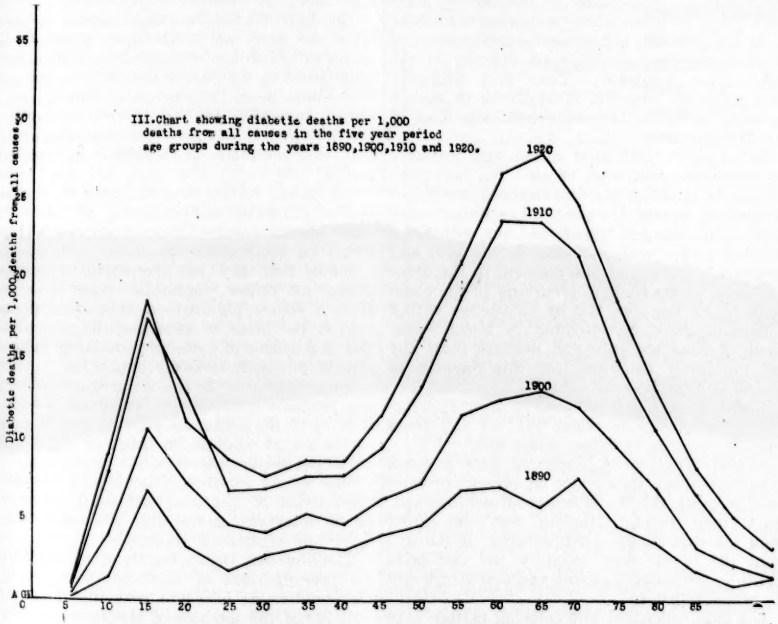
DIABETIC MORTALITY AS COMPARED WITH GENERAL MORTALITY

The most important fact in this graph is the significance of the period of life from five to fifteen years with its marked increase in diabetic deaths as compared with the general mortality during this same period.

In the curve for 1890 we find, that compared with all other causes of death, diabetes was almost as common a cause of death at fifteen years as at seventy. Thus at the age group ten to fif-

teen years in 1890, out of every 1,000 deaths from all causes there were 6.89 due to diabetes. At sixty-five to seventy years of age of every 1,000 deaths from all causes 7.64 were due to diabetes. In other words during the period ten to fifteen one death out of every 143 deaths was due to diabetes, while at sixty-five to seventy one out of every 125 deaths was a diabetic death. It will be noted that this is characteristic of the curves for the other three years, but is however less marked in each of the succeeding decades.

The relatively increased diabetic mortality, that is diabetic deaths per 1000 deaths from all causes, begins at about forty years and reaches its maximum on an average at about sixty-five years when it rapidly falls, approaching zero at ninety to ninety-five years. This is somewhat in contrast with the diabetic mortality curve where the rise begins at forty-five and reaches its maximum at seventy-five to eighty years.



COMPARISON OF GRAPHS

In comparing the diabetic mortality curves with those of general mortality it is found that in the diabetic mortality the rise begins at forty-five to fifty years whereas the general mortality rates show no considerable rise until sixty-five to seventy years of age. It is also significant that in the diabetic mortality curve the height occurs at seventy-five to eighty while in the general mortality curve the rise is progressively upward toward absolute mortality. This maximum occurring in the diabetic curve is easily explainable by the fact that the more severe diabetic has succumbed before seventy-five years has been reached, while the majority of cases occurring or persisting after this age are of the more mild type and their length of life as compared with general mortality is not markedly influenced by the disease.

The marked increase at ten to fifteen years of diabetic deaths per 1,000 deaths from all causes is not due to an actual increase of diabetic deaths occurring during this period but is entirely a relative affair as there is no appreciable rise in diabetic mortality during this period. There is however a very definite decrease in the general mortality which combined with the general upward trend of the diabetic curve gives a relative rise in diabetic mortality as compared with the general mortality curve for the same period.

SUMMARY

The general mortality in the registration area of the United States for the four decades between 1890 and 1920 shows a tendency to decrease which is greatest between the age five to ten years and next greatest for the ages between ten and fifteen. For these same decades, as well as for the two five year age period above mentioned, the diabetic mortality shows no decrease but rather a gradual rise. In consequence the

diabetic deaths per 1,000 deaths from all causes show a marked increase between 1890 and 1920.

In comparing the diabetic mortality with the general mortality it is manifest that the diabetic mortality reaches two distinct elevations, a peak and a plateau. The first of these is the peak and occurs at the age of fifteen years and is a relative increase while the second, the plateau, develops between sixty and seventy years of age and is an actual increase,

The diabetic mortality curve begins to rise rapidly at from forty-five to fifty years of age, reaching its maximum at eighty years for all four decades and then rapidly falls.

HYPERBILIRUBINEMIA IN DISEASES OF THE BILIARY TRACT

BY MICHAEL F. FALLON, M.D., WILLIAM F. LYNCH, M.D., AND JOHN J. DUMPHY, M.D.

ANY test which will give accurate results in the diagnosis of diseases of the biliary tract must prove of great value and more especially to the surgeon who is frequently embarrassed in determining the operability of diseases in the right upper quadrant. Time and authority have given the sanction of approval to such a test—the so called Hyperbilirubinemia Test of van den Bergh.

McNee says: "the most recent and probably the most important work of all" (on jaundice) "is due to Hijmans van den Bergh, formerly of Groningen, now of Utrecht. The writer more than anyone else has transferred our outlook on jaundice away from the color of the skin and presence or absence of bile pigment in the urine and feces, to the changes occurring in the blood serum. He has done this by his success with a method of detecting bilirubin in blood serum which is more accurate and delicate than any test previously employed for this purpose in clinical or experimental work."

Rowntree says that the status of liver functional tests today is about that of the renal function test ten to fifteen years ago.

Of the various liver functional tests, a dozen or more, the two tests which at present promise definite value are the Rowntree-Rosenthal test, the Phenoltetrachlorphthalein test, depending upon the rate of the disappearance of the dye from the blood; and secondly the test with which we are concerned, the van den Bergh test for serum bilirubin.

The knowledge that bile coloring matter is in the blood is nothing new, since even Malpighii used the crude method of dipping a cloth in the blood, and if the color of the cloth became yellow, he inferred jaundice.

The old theory that the bile pigment, bilirubin, was secreted in the liver has been revolutionized. Within the past few years it has been definitely proven that bilirubin in whole or in part is secreted outside the liver. The most striking evidence of this comes from the studies of Whipple and Mann. The latter showed after the liver has been removed completely from animals there was still a progressive accumulation of bilirubin in some of the tissues, mostly in the blood. As a result of these investigations we must regard the liver as an excretory rather than a secretory organ for bilirubin. And this new knowledge changes radically our ideas about jaundice.

The bilirubin in the blood serum is chemically the same as in the gall bladder. The amount of bilirubin normally in the blood serum is about 0.3 to 0.5 part to 200,000. Visible jaundice occurs when the amount of bilirubin in the serum increases to four parts or more in 200,000; and it is only after this concentration that the excretion of bilirubin in the urine occurs.

The blood serum owes its color to the presence of bilirubin, and ordinarily bilirubin is the only yellow pigment present in the human serum in appreciable quantity. Occasionally a yellow tint may be present from excessive eating of yellow vegetables, such as carrots. Here a yellow pigment—a lipochrome pigment —as in the blood of cows may be present; but this as a source of error in estimating the quantity of bilirubin is easily controlled.

Since bilirubin is the only appreciable yellow pigment ordinarily in the blood, and since it gives to the serum its yellow color, the depth of the serum color is an index to the quantity of bilirubin present.

The serum color is estimated by measuring the number of dilutions required to render the serum color just perceptible when viewed in a layer one centimeter thick.

The van den Bergh test is a confirmation of the investigations of Ehrlich and is based on the coupling of bilirubin with diazonium salts.

It is not the purpose of this paper to discuss the significance of the direct and indirect reactions of the van den Bergh test. Rather is it the object to call attention to the reliability and simplicity of the test, and to communicate some of the results that may be definitely expected from it as reported by some investigators and from our own experience with it.

(There is at present more or less confusion as to the exact significance of the direct and indirect reactions. The direct reaction is immediate after the addition of the diazo reagent; the indirect, after the addition of the diazo reagent and alcohol. The difference is based on the belief that the bilirubin of the bile is not exactly the same as the chemically pure substance.)

While the increase of bilirubin in the serum

will show progressive jaundice, it does not tell to what the jaundice is due; since the jaundice may be due to blockade or infection in the biliary tract, or even to passive congestion of the liver.

It is generally accepted that the van den Bergh test shows the presence of jaundice whether arising from infection, inflammation or toxaemia; and since this is true, the possibilities of its being an aid to the surgeon in diagnosis and in treatment are many. For example, the presence of an increased quantity of bilirubin in the serum, before visible jaundice occurs, may aid in differentiating surgical diseases of the biliary tract from the other surgical dyspepsias, cancer and ulcer of the stomach, ulcer of the duodenum and appendicitis; in all of which the amount of bilirubin may be normal.

Again, in some cases, when it is difficult to decide whether the lesion is in the right kidney or in the biliary tract, the presence of an excess of bilirubin in the serum may be of aid in diagnosing the disease to be in the biliary tract. Since the bile goes into the blood first, and leaves the blood last, the Icterus Index shows whether the jaundice is increasing or receding or at a stand still. Hence it may be of great aid in determining the proper time for operative interference; and in post-operative patients it may indicate the presence or absence of trouble with the biliary apparatus, even when the clinical signs do not show it.

The test gives an accurate estimate of the intensity of jaundice, whether it be absent or present in the skin, sclerae or urine. It has already proven of value by indicating beginning damage to the liver by toxaemia resulting from the treatment of syphilis.

The Icterus Index is also an aid in establishing the presence of jaundice in dark skinned races, such as Negroes and some Orientals.

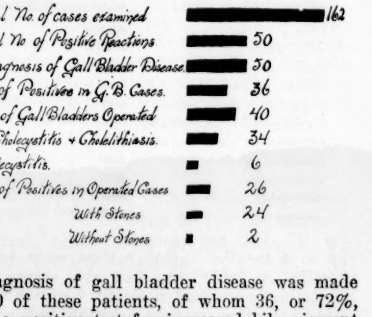
The test will indicate all that visible jaundice would indicate, but even more than that since it gives an accurate estimation of the intensity of the jaundice and since it will tell of its increase or decrease; moreover, there are no known tests to quantitate icterus by the skin or urine.

The van den Bergh test has, by competent observers, been proven so reliable as a quantitative test of the serum bilirubin, that it is entitled to a place in the laboratory as a routine measure in diagnosis and prognosis, especially in diseases of the right upper quadrant of the abdomen. Also, in as much as it is dependable in calculating the degree of icterus, the test when further developed promises to be even more valuable than it is at present.

Dr. C. H. Green of the Mayo Clinic, who has done excellent work on this test, says: "In experimental obstructive jaundice, we have found that a very close parallelism between the serum bilirubin and phenolotetrachlorphthalein retention could be demonstrated; a fact which demonstrates the close relationship between the degree of icterus and hepatic insufficiency."

Here follows our experience with Hyperbilirubinemia in Diseases of the Biliary Tract.

The bloods of 162 of the surgical patients entering St. Vincent Hospital from May 1, 1924, to September 15, 1924, were examined for Hyperbilirubinemia by the methods of van den Bergh and of Faucher. All cases of suspected gall bladder disease were included in this group, the others being unselected and taken for controls.



Diagnosis of gall bladder disease was made on 50 of these patients, of whom 36, or 72%, gave a positive test for increased bile pigment in the blood by one or both of the above methods. Of the 50 patients with diagnosis of gall bladder disease, 40 were operated; 39 cholecystectomies were done and one removal of stone from common duct on a patient with a previous cholecystectomy.

Of these 40 patients on whom operation was done 33 had no visible jaundice, 4 had visible jaundice and 3 were doubtful. 26 of these 40 operated cases gave a positive test—65%. In 34 of the 40 cases, gall stones were found at operation; of these 34, 24, or 71%, gave a positive reaction. There were 6 cases of cholecystitis without stones, of which 2 gave a positive reaction which disappeared after cholecystectomy. Of the 26 cases giving a positive reaction and clinical evidence of gall bladder disease, who were operated, stones were found in 24.

Of the whole 162 cases on whom the test was done 50 positives were found. Of them 36 were proved or suspected cases of cholelithiasis. The other 14 included 2 cases of definite jaundice, one from cirrhosis of the liver and the other from a retroperitoneal tumor; 2 cases of slight jaundice with severe anemia giving delayed van den Bergh reactions; 2 cases of pelvic peritonitis; 1 case of varicose ulcer; 3 cases of appendicitis; 1 case of cardiac failure; 1 case of endometritis; 1 case of hemorrhoids and 1 case of migraine.

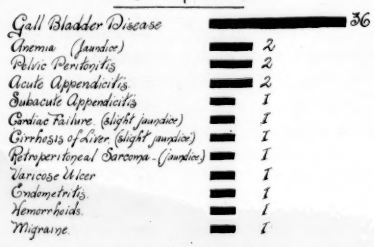
Among the negative cases were 10 cases of appendicitis, 6 cases of duodenal and 2 cases of

gastric ulcer, and 7 cases of cancer of the gastro-intestinal tract, all of which were operated.

We believe the reaction is especially valuable in differentiating acute attacks of abdominal pain due to gall bladder disease. In 20 successive cases coming to operation 14 had acute attacks of gall bladder disease within one week of entrance and every one of these gave a positive test. The remaining six were negative. The shortest interval from an attack in these negative cases was 9 days and the others varied

(2)

Positive Reactions



up to 4 months. One patient came into the hospital in labor who had also severe upper abdominal pain and repeated vomiting. Her blood showed a positive reaction and although she was not operated, subsequent examinations showed the diagnosis of gall stones to be reasonably certain.

A certain number of positive reactions were obtained in other conditions and one might suppose they would be confusing. However, of the 50 positive reactions in only one case did it contribute to the wrong diagnosis and in only

(1)

Proved Diagnosis	No. of Cases	Positive	Negative
Cholecystitis	40	26	14
Duodenal Ulcer	6	0	6
Gastric Ulcer	2	0	2
Appendicitis	13	3	10
Cancer (G.I. Tract)	7	0	7
Empyema	4	0	4
Rt Pyonephrosis	1	0	1
Lung Abscess	1	0	1
Lead Poisoning	1	0	1

3 cases of acute abdominal pain other than gall bladder disease was the test positive. Two of these cases were acute gangrenous appendices in which the diagnosis was evident.

A remarkably interesting fact was observed in doing these tests. That is, that all bloods taken following etherization proved to be positive. A series of cases was done in which the test was performed both before and after ether; and in all cases except two very short etherizations, they became positive following ether. This positive reaction subsequently disappears. This fact is interesting in the possibility of ether damage to the liver and is being studied further.

SUMMARY

Of 50 gall bladder cases, 36 or 72% gave a positive test for Hyperbilirubinemia. Of 40 cases of diseased gall bladder coming to operation 26, or 65%, gave a positive test for Hyperbilirubinemia.

Of 26 positive tests with symptoms of gall bladder disease 24, or 95%, had stones.

Of 20 successive gall bladder cases operated, 14 had attacks within a week and all of these had positive tests.

Patients after etherization almost always show a positive test.

CONCLUSIONS FROM OUR EXPERIENCE

1. Detection of Hyperbilirubinemia is of definite value in the diagnosis of gall bladder disease if interpreted in the light of other findings.
2. A positive test will rarely lead toward a wrong diagnosis.
3. It is of especial value in acute cases; and a positive reaction will usually be obtained within a week of an acute attack.
4. It is decisive in cases of doubtful jaundice.
5. Etherization causes Hyperbilirubinemia.

TECHNIQUE

There are several modifications of the technique of the van den Bergh test, all of which give satisfaction to those who have acquired experience in their use. The following plasma method was recommended by Dr. Broun of the Boston City Hospital and has proven satisfactory as qualitative method to determine the presence or absence of Hyperbilirubinemia.

(1) 1 Drop of 20% Potassium Oxalate. 5 c.c. Blood, mix gently. Centrifuge—Remove plasma.

(2) To 1 c.c. plasma add 2 c.c. distilled water—mix gently. To this mixture add 0.8 c.c. of freshly prepared diazo reagent.

DIAZO REAGENT

Solution 1.

Sulphanilic Acid 4.5 grams
Concentrated Hydrochloric Acid 50 c.c.
Distilled Water to make 500 c.c.

Solution 2.

Sodium Nitrite 2.5 grams
Distilled Water q.s.—500 c.c.
"Just before use—mix 50 parts of No. 1.
1 part of No. 2."

After mixing the diluted plasma with the diazo reagent watch the tube; preferably against a white background. If a distinct purple color develops within 1 minute the reaction is prompt—and indicates liver pathology.

If the reaction occurs later than 1 minute—(usually within 15 minutes) the reaction is delayed and indicates the presence of jaundice of hemolytic origin.

If no purple color develops at all the amount of bilirubin in the plasma is less than 1 mg. per 100 c.c. which means that there is no jaundice or only slight jaundice.

Normal individuals have as much as 0.5 mg. bilirubin per 100 c.c. of blood plasma, and a few may have more than this particularly when fasting.

As a quantitative determination of the degree of bilirubinemia the following serum determination of bilirubin is used in the laboratory of Dr. Rowntree at the Mayo Clinic. (Thannhauser and Anderson Modification of the van den Bergh Method.)

1. Principle.

Sulphanilic acid and sodium nitrite when added to solutions of bilirubin, form a colored addition product azo-bilirubin.

2. Solutions.

1. Standard artificial bilirubin solution.
   a. Dissolve .1508 gms. of ammonium iron alum in 50 c.c. of concentrated hydrochlorie acid and add water to make 100 c.c. Keeps indefinitely.
   b. To 10 c.c. of solution a add 25 c.c. of concentrated hydrochlorie acid and water up to 250 c.c. Keeps about 6 months.
   c. The standard for comparison with the unknown is made by adding to 3 c.c. of (b) an equal volume of 20% potassium sulphocyanide and 12 c.c. of ether. Shake well and transfer ether extract to colorimeter cup. Must be prepared fresh each time. This solution corresponds in color to a 1 in 200,000 solution of azo-bilirubin, or 0.5 bilirubin per 100 c.c.

2. Sulphanilic Reagent.

This is a freshly prepared Ehrlich's diazo reagent made of two solutions each of which keeps well, but the mixture must be made immediately prior to the test.

Solution A.

Sulphanilic acid 5 gm., concentrated hydrochlorie acid 50 c.c., distilled water to make 1,000 c.c.

Solution B.

Sodium nitrite 5gm., distilled water to make 100 c.c. Mix A and B in proportion of 0.8 c.c. of solution B, made up to 25 c.c. with solution A.

3. Saturated ammonium sulphate.

4. 95% ethyl alcohol.

3. Procedure.

To 2 c.c. serum, add 1 c.c. of the freshly prepared sulphanilic reagent. The color change at this point permits the reading of the test in the terms of direct or indirect reaction. 2 c.c. of saturated ammonium sulphate are added to help precipitate the serum proteins, followed by 10 c.c. alcohol. The tube is centrifuged and the supernatant fluid compared colorimetrically with an ethereal solution of ferric-sulphocyanide prepared according to the directions of van den Bergh. The color quality of the two solutions is not always identical and other artificial color standards have been advised. In general, the above technique has been satisfactory.

Calculation.

$$\frac{\text{Reading of standard}}{\text{Reading of unknown}} \times 3.75 \text{ equals mg. bilirubin in 100 c.c. serum}$$

Normal blood contains less than 2 mg. bilirubin per 100 c.c. serum.

Example:

$$\frac{\text{Standard at 20}}{\text{Unknown at 10}} \times 3.75 \text{ equals 7.5. mg.}$$

$$\frac{\text{Standard at 3.}}{\text{Unknown at 20}} \times 3.75 \text{ equals .56 mg.}$$

It will be noticed that the strength of the sulphanilic acid solution is different in these two tests and it is the writers' experience that the stronger solution as used in the former gives better results, probably because of variation in the quality of sulphanilic acid.*

*In preparing the standard solution care should be taken to select only clear violet crystals of ammonium iron alum. This test is probably more suitable for determinations in research work than as a routine procedure. In our experience the former qualitative method is satisfactory for clinical use.

The Fauchet Oxidation test is as follows:—
Reagent consists of—Trichloracetic Acid—5 c.c.
Ferric chlorid solution 10%—2 c.c.
Distilled water—20 c.c.

This test is carried out by adding from 3 to 5 drops of this solution to an equal amount of serum.

If bilirubin is present to an extent of 1-60,000 or over, a green develops. This color change is a specific for bile pigment.

In our experience the Fauchet test is less delicate and in general less satisfactory than the van den Bergh reaction. The end point is often not sharp and difficult to interpret.

The fact that in a few cases the Fauchet test is positive while the van den Bergh is negative

is explained by Friedman and Straus as probably due to the oxidizing process of the Fauchet reagent breaking up combinations of bilirubin which would not otherwise react.

In all of these tests care must be taken to avoid the slightest hemolysis.

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THE CLINICAL APPLICATION OF RECENT STUDIES IN JAUNDICE*

BY SAMUEL C. HARVEY, M.D., NEW HAVEN, CONN.

JAUNDICE since the time of Virchow has had a fascinating interest for the pathologist and the clinician alike. From the first of the modern work it was recognized as a condition due to the presence of an abnormal amount of bilirubin in the blood stream, which found its way into the other tissues of the body, producing the characteristic discoloration, and penetrating through the kidneys into the urine, where its presence ensured the diagnosis. In certain instances, as in hemolytic jaundice, bilirubin is not found in the urine and this is called "acholuric jaundice." In other cases the pigment is not accompanied by the bile salts as it is in the obstructive type and this is spoken of as a "dissociated jaundice." And lastly, with the introduction of the more delicate and quantitative methods for the determination of bilirubin in the serum, its presence may be detected in more than the normal quantity (for there is always a certain amount present), without discoloration of the skin or sclerae, and this is termed "latent jaundice."

To provide an hypothesis which will explain all these derangements of bilirubin formation and excretion has taxed the ingenuity of many investigators. It has always been obvious that the fundamental question was that of the site of the formation of bilirubin.

As early as 1840-50, Johanes Muller, Kunde and others extirpated the livers of frogs and as long as four days afterward found no bile pigment in the blood. These experiments were shown to be inconclusive, however, as tying the common duct in this animal does not produce jaundice, in such a short period of time. From an histological study of the liver, with particular reference to the distribution of bile pigment within it, Virchow became convinced of the correctness of the prevailing opinion that bilirubin was formed in the liver. Hoppe-Seyley and others in the meantime identified hematoidin and bilirubin as being the same substance; and Virchow found hematoidin in old hemorrhagic exudates in the form of crystals which gave the chemical reaction for bilirubin. The latter then assumed that in addition to the liver as the originator of bile pigment, one must include other tissues, and formulated his dual hypothesis of the hepatogenous and the anhepatogenous origin of jaundice. This remained the orthodox teaching for many years, to be specific until 1886, when Minkowski and his pupils attacked the anhepatogenous or extra-hepatic origin of bile pigment by means of a series of cleverly devised and well carried out experiments on birds. In these animals it is possible to remove the liver with relative case and with their survival for the necessary length of time, a thing that, until the recent work of Mann, it has been impossible to accomplish in any higher animal than the bird. Minkowski found that following the removal of the liver no bilirubin could be detected at any time in the blood. Moreover, this still remained true when intensive hemolysis was produced by arsenurated hydrogen. This was universally accepted as conclusive proof, and Rolleston (as late as 1912) states in his textbook of diseases of the liver that there is little or no evidence of the formation of bilirubin elsewhere than in the liver. Since Minkowski's experiments most observers have been busily at work forcing their findings to conform to the supposedly established fact that bilirubin is formed in the parenchymal cells of that organ. Thus, Stadelman, whose compendious monograph, "Der Icterus," is a model of thoroughness and completeness, made the important observation that *toluylenediamine* produced hemolysis *in vivo*, and with it jaundice. He also noticed that there was extensive damage to the parenchyma of the liver, and that the bile was thick and viscid. He reasoned then that the parenchymal cells, laboring to turn the excess hemoglobin into bilirubin, produced such a quantity that the biliary capillaries were plugged and in a similar manner as in the obstructive jaundice caused the damage seen in the liver cells. Eppinger in cases of "hemolytic jaundice" found "bile thrombi" in the biliary capillaries, and, reasoning in the same manner as Stadelman, supposed obstruction and a damming back of bile into the circulation. In other words, if bilirubin is formed by the liver cells, then it can only appear in the circulation by a reversal of its flow

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from the cell, which is most readily supposed on the basis of obstruction of one form or another.

It is now of interest to note that Minkowski in 1900 described in detail a disease entity, "hemolytic jaundice," in which increased fragility of the red cells, splenomegaly and low grade jaundice without any demonstrable liver disease, suggested to the minds of many that Virchow's supposition of an anhepatogenous jaundice might be correct. In 1911 the spleen in such a case was removed by Micheli with a cure and the same year Pearce and his collaborators studied experimentally the relation of the spleen to blood destruction and the production of bilirubin, and found suggestive evidence that this organ played a large role in bile pigment formation.

A similarly suggestive line of work was that of Aschoff and his pupils, who, following observations of Ribbert made in 1904, studied certain large endothelial cells with a phagocytic function which are found in the walls of blood spaces in various portions of the body. Notable examples of these are the endothelial cells in the spleen and the Kupfer cells in the liver. In an extensive blood destruction these cells take up the corpuscular debris and hemoglobin, and soon becoming loaded with hemosiderin, the iron containing moiety. This suggested that the liberation of the organic component, which is bilirubin or its precursor, might well take place in these same cells.

McNee, who was working with Aschoff in 1911, confirmed the observation, that in the bird a great majority of the cells of this type lie in the liver, the spleen being a relatively small organ. Supposing these cells, then, to be concerned in the production of bilirubin, extirpation of the liver of the goose as was done by Minkowski would remove the greater part of this tissue as well as the parenchymal cells. It follows, then, that the experiments of Minkowski which had for so many years been the mainstay in the argument for the hepatic origin of bilirubin are invalid and of little or no significance. If one could remove the liver in a mammal where the majority of the endothelial cells lie outside of that organ one might arrive at some conclusion.

In 1911 Whipple attempted to set up this type of an experiment. He deviated in the dog a large part of the blood stream away from the liver by throwing the portal stream into the inferior vena cava through an Eck fistula. There was no appreciable diminution in bilirubin formation. He also shut off the lower part of the body, including the liver, from the circulation and on injection of hemoglobin in the upper half, obtained bilirubin in the serum. These experiments, because of the technical difficulties into which I cannot enter, have not been generally accepted as conclusive, but they have received, very recently, confirmation in the work of Mann. This investigator, by an ingenious three-stage procedure in dogs, has deviated the portal circulation and completely removed the liver. These animals, with suitable care, live from twenty-four to thirty-six hours and from the sixth hour onward become progressively more deeply jaundiced. The proof is then definite and conclusive that bilirubin is formed elsewhere than in the liver, and there remains no similarly conclusive proof that it is formed by the parenchymal cells of the liver.

Van den Bergh, whose monograph, "Der Gallenfarbstoff im Blute," reflects the newer view, in an effort to find a delicate test for bilirubin in the serum availed himself of the reaction described by Ehrlich in which a diazonium salt couples with bilirubin to give a salt, azobilirubin, which has a brilliant color. In "obstructive jaundice," when the reagent is added directly to the serum, the reaction takes place at once, while in hemolytic jaundice it is usually necessary to extract first with alcohol. This has led Van den Bergh to suppose two kinds of bilirubin in the serum, one of which has passed through the liver cells and then been returned to the blood stream as in the obstructive type, while the other has never left the blood stream, as in the hemolytic type. That, in the face of obstruction, bilirubin should still pass through the liver cells seems improbable, at least it is a supposition not to be made without further proof. I feel, with Thannhauser and with Brulé, that it is probably a matter of the absorption of bilirubin by the colloid constituents of the serum when the pigment is a long time in contact with them and that the extraction with alcohol breaks down this physical coupling. There is then even here no strong evidence of any participation of the liver cell proper in the production of bilirubin.

The function of the liver cells, so far as the evidence at hand goes, is merely that of a filter, analogous to the action of the kidney cells in excreting urea. If the cell is damaged, this filtration action is interfered with and there is a damming up of bilirubin in the circulation. The capacity of the liver cell for the excretion of bilirubin is great. We know from Stadelman, Whipple and others as well as from experiments done in our own laboratory that the injection of hemoglobin leads to a marked increase in the output of bile pigment as measured by means of a biliary fistula. In dogs, at least, it is difficult to increase the production of bilirubin by hemolysis to a point where the liver will not excrete it as rapidly as it is produced. This we have ascertained by injecting large quantities of hemolyzed blood and testing the serum for bilirubin over a considerable length of time.

On the other hand it is easy to produce experimentally an intense jaundice by the use of substances which do injury to the liver cell without producing hemolysis. An example of

such an agent is chloroform. If a biliary fistula is established in the dog by means of a tube and rubber balloon as described by Rous and McMasters one can collect quantitatively the output of bile. There is at first a depression of the output, which on the third day returns to normal. If at this time the animal is injected with chloroform, we know that a definite and precise damage is done to the parenchymal cells of the liver. At once the biliary output is decreased in a proportionate amount to the injury done. Bilirubin appears in considerable quantities in the blood serum as shown by the Van den Bergh reaction and overflows into the urine. If the damage is sufficiently severe the animal becomes outspokenly jaundiced. When the liver recovers, as it will with a relatively slight injury in three to four days, the bilirubin output increases to above the normal amount and the urine and serum become clear of the pigment. With a lighter degree of damage, but sufficient to show all these phenomena, so that without question the injury is confined to the parenchymal cells, there are no "biliary thrombi of Eppinger," and no evidences of obstruction. There is no lack of production of bilirubin to account for this phenomena as is shown by the appearance of pigment in the serum and in the urine. This combination of facts indicates rather clearly, I believe, that in the dog, jaundice or hyperbilirubinemia means decreased liver function.

There are three major methods of escape of bilirubin from the blood stream: the liver, the body tissues and the kidneys. It is apparent from the work of many investigators that in man there is a normal bilirubin content of the serum, of about one part in 200,000, and that any amount materially above this will be passed out through normal liver cells into the bile passages. This represents the threshold of excretion of bilirubin by the liver. If this threshold is raised then the pigment begins to accumulate in the tissues. It is probable, that because of its low solubility it does not rapidly pass into the protoplasmic menstruum of the cell but enters slowly in, by a process of colloidal absorption. For the same reason, its discharge from the cellular structures is likewise slow and it is prone to be precipitated as granules of pigment within the cell itself. For this reason there is a certain lag in the appearance and disappearance of jaundice of the tissues as compared with the bilirubin content of the blood. Given time, however, the two probably adjust themselves to the same level, but if the elevation is relatively slight, that is one part in 80,000, as compared with the upper normal limit of one in 200,000 then there is a jaundice, so to speak, which does not produce sufficient pigmentation of the skin and sclerae to be recognized by the eye. It is important then that the clinician, in suspected liver dysfunction, make observations on the bilirubin content of the serum.

There is a third major route of escape for the bilirubin of the serum and that is through the kidney. The cells of this organ are, as compared with the liver or the tissues in general, relatively impervious to bilirubin, the threshold for overflow being a concentration of about one part in 50,000. It is apparent, therefore, that the qualitative tests for bile in the urine are rather crude and uninstructive as compared with the determination of the bilirubin content of the serum.

What is the relation of the points brought out in this discussion to the clinical entities in which jaundice appears? Let us take first the ordinary "obstructive jaundice." A stone blocks the common duct, and bile is secreted through the parenchymal cells of the liver until the pressure in the ducts and the concentration of the bile reaches a point where the cell can no longer function. The bilirubin formation is going on elsewhere in the body, is not excreted through the liver, and accumulates in the blood giving an hyperbilirubinemia. When this reaches a concentration of about one in 80,000, the skin and selerae become jaundiced and at one in 50,000 bile appears in the urine. The accumulation continues until the kidneys secrete it as rapidly as it is formed and in that way a concentration as high as one point in 4 or 5,000 in the blood may be reached. The process is analogous to that which takes place when there is a block in the ureter with the loss of the ability of the kidney cells to excrete urea and its consequent accumulation in the blood.

Let us consider the ordinary catarrhal type of jaundice. There is an infection of the biliary tract which ascends until it involves the parenchyma of the liver, with a consequent dysfunction as regards the excretion of bilirubin. The same sequence of events as in the preceding example takes place and we have jaundice. This may be compared with a pyelitis and a pyelonephritis with consequent retention of urea.

If we consider the infections and intoxications attacking the parenchymal cells themselves as "icterus spirochetose," acute yellow atrophy, chloroform poisoning, "salvarsan jaundice," etc., the explanation is the same. In profound infections elsewhere in the body accompanied by jaundice, as in pneumonia and septicemia, there is in all probability, as Brulé suggests and the French school believes, a profound damage to the parenchymal cells of the liver and interference with their function. In metastatic carcinoma of the liver, in cirrhosis and in syphilis of the liver a similar parenchymal damage explains the accumulation of bilirubin in the blood. Only in the "hemolytic jaundice" and in pernicious anemia where there is a slight but distinct hyperbilirubinemia is there no demonstrable liver lesion, and it may well be that these diseases involve the formation of a pigment closely related to bilirubin, for which

the threshold of excretion of the liver and kidney is higher than with that pigment itself.

It is apparent that the clinician, with this concept in mind and with the newer methods of detection of bilirubin in the blood serum at hand, must revise his ideas of jaundice and of its relationship to disease. It is now possible to ascertain at an early moment and to interpret intelligently any damage to the liver parenchyma which affects the excretion of bilirubin.

It is of interest to note briefly such findings in a large and varied group of diseases. The author is indebted to Dr. Edward P. Levine for the data herewith presented, which was obtained in the clinic of the New Haven Hospital and presented as a thesis for the degree of Doctor of Medicine, Yale University, 1924.

The icteric index of the serum was determined by the Maue modification of the Meulengracht quantitative test. It is carried out as follows:

Approximately 5 c.c. of blood are withdrawn from the vein. This is allowed to clot and then it is loosely separated from the wall of the test tube and the blood centrifugalized. The serum is compared in a colorimeter against a standard potassium chromate solution of a strength of 1-10,000. The quotient obtained by dividing the reading of the standard by the reading of the unknown gives the icteric index. We have taken any reading above 18 as representing a definite hyperbilirubinemia. Readings below this may be suggestive but not conclusive. The sera of 300 patients were investigated by this method. In 287 of these the reading fell below the level considered as definitely abnormal; 104 of these normals lay in the following general groups:

Rickets	6
Pregnancy	22
Syphilis	31
Diabetes mellitus	4
Tuberculosis	11
Peptic ulcer	7
Carcinoma of esophagus	4
Acute cholecystitis	4
Pernicious anemia	2
Pneumonia	13

The remainder of this normal group are distributed in a miscellaneous fashion through a great variety of diseases. Those patients showing abnormal readings are as follows:

Varicosities 1—(28)
Infectious jaundice 1—(105)
Common duct stone 3—(60-155-160)
Hemolytic jaundice 1—(42)
Influenzal jaundice 1—(31)
Metastatic carcinoma of the liver 1—(159)
Carcinoma of the pancreas 1—(160)
Cholelithiasis 3—(88-100-153)
Pneumonia 2—(22-42, both died)

It is apparent that the simple determination of an icteric index alone, by what is a relatively rough method, affords considerable information. It enables one to detect the latent type of jaundice, and leads one, even without a great rise in the index, to suspect a liver injury in instances where it would be otherwise overlooked. It is also of definite value in the prognosis in patients suffering from acute infectious disease, such as pneumonia, where an elevated index suggests an unfavorable outcome. It is of particular value to the surgeon in differentiating between disease of the liver and bile ducts and disease of the stomach, duodenum, appendix, etc. It is also of considerable help in following the progress of an outspoken jaundice, as the increase or decrease of the bilirubin in the blood serum can be determined some time before the changes in the urine and tissues are very apparent.

Conclusion

The newer concepts of the formation and excretion of bile have tremendously simplified our understanding of the mechanism of jaundice and by directing our attention to the bilirubin content of the serum, rather than to that of the skin or urine, have enabled us to detect the presence and follow the course of this symptom with greater accuracy and with a more complete interpretation of its significance than ever before.

A STUDY TO DETERMINE THE PRESENCE OF AN OSTEOPATHIC LESION IN DISEASE OF RECOGNIZED PATHOLOGY*

BY WILLIAM P. MURPHY, M.D., AND PERRIN T. WILSON, D.O., BOSTON

The policy and duty of the physician of today is to make use of every proved therapeutic method which offers relief from suffering or cure for disease. Many therapeutic methods have been formulated and tried out since the beginning of medicine. Perhaps the majority of these have been discarded in whole or in part because of lack of proof that their underlying principles are sound or that they have offered a rational addition to the therapeutic measures of the time. Many of these methods are still under careful observation and each year facts are established by means of careful research which enable us to add to our therapeutic assets or to discard ill-founded and unsatisfactory methods.

*From the medical clinic of the Peter Bent Brigham Hospital. This work was undertaken as part of an investigation that has been conducted on the subject of Osteopathy and Chiropractic by a committee of the Council of the Massachusetts Medical Society. The committee consisted of Channing Frothingham, M.D., Chairman, George S. C. Badger, M.D., and James W. Sever, M.D.

As our system of therapeutics becomes more and more stable through scientific observation and control, therapeutic measures based on empiricism and misdirected enthusiasm are not accepted by the more enlightened and conservative of the medical or lay populace until proof of their sound basis or of their therapeutic value has been presented.

The enlightened and conservative element of the medical and lay populace becomes more numerous each year as more interest is centered on medicine through improvement in results of treatment and through intelligent publicity of facts concerning the diagnosis and treatment, as well as the symptoms of disease.

The same type of scientific investigation which is being employed to substantiate or to improve the therapeutic measures accepted by the medical profession today should be employed to determine the soundness of the basic theory and the therapeutic value of those therapeutic suggestions which are as yet not generally accepted by the medical profession.

During the past fifty odd years osteopathy, as a therapeutic method, has been struggling for recognition. The principle of treatment by the osteopathic method was first formulated in 1874 by Dr. A. T. Still, at that time a regular practicing physician. His original idea was largely empirical but his later observations are reported to be the result of "careful anatomical investigation." In spite of the fact that Dr. Still is said to have carried on anatomical investigation and that years have been spent in an effort to establish the theory and practice of osteopathy; there is little to be found in print which is of the nature of consistent or controlled investigation which would convince the more discerning physician or lay citizen that the original hypotheses were sound or that treatment by this method accomplishes that which is claimed for it.

If there is value in osteopathic treatment then this should be recognized through proper investigation. If this value is in the form of another useful therapeutic method, it should be available for the use of the physician through the regular channel, the standardized medical schools of the country or through men well trained in the principles of this method who are willing to cooperate with the physician. For these reasons it seemed advisable to attempt some careful work along this line in the wards of the Peter Bent Brigham Hospital.

Two main problems presented themselves for solution. The first was to attempt to demonstrate the presence of an osteopathic "lesion," while the second problem was to observe the effect of adjustment of the "lesion," if such were found to exist. The work summarized in this paper deals only with the first of these problems.

An osteopathic lesion is defined as any maladjustment of structure which results in perverted function (disease). In order to search for an osteopathic lesion it would be necessary to assume or prove a relation between maladjustment of structure and perverted function (disease). As such a problem has not been solved in respect to the vertebral column as yet and is obviously a difficult one to solve, the present problem was limited to an attempt to demonstrate a constant structural maladjustment for any given type of perverted function or disease.

The recognition of structural maladjustment in general is not an observation limited to the practice of osteopathy. The recognition of structural maladjustment along the vertebral column is a more distinct and basic element of the osteopathic principle so an effort was made to demonstrate structural abnormality in this region only.

Structural maladjustment such as might be concerned in a "lesion" may consist in any one or more of the following changes:—

1. Muscle spasm.
2. Tenderness or pain.
3. Abnormal motion.
4. Pigmentation.
5. Bony maladjustment.

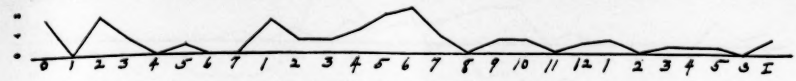
The maladjustment most frequently recorded was a bony one. In the early part of the work tenderness and muscle spasm were recorded. These were found to be very inconstant and in some cases so extensive that little could be accomplished along that line so they were not recorded as such. An attempt was made, however, to record them wherever they were found in conjunction with a bony abnormality.

In most of the cases an examination of the vertebral column was made by two observers without previously knowing the diagnosis except as this might be very evident, e. g. in the asthmatics. Each man recorded his findings independently. In this way each man's results were affected as little as possible by that of the other or by preformed opinion. The patients examined were in most cases in condition so that they could sit up with ease in bed and so be manipulated as desired in order to examine and demonstrate the structural changes. The diagnosis used in each case is that recorded in the records of the Peter Bent Brigham Hospital.

It is evident from the foregoing discussion that in order to demonstrate the sound basic principle of osteopathy it is necessary to demonstrate structural maladjustment which is constant at a certain vertebral level in a patient suffering from a given perversion of function (disease) and which should be present in only a small percent of patients suffering from other perverted functions (diseases).

OBSERVATIONS

The following report is based on the examination of 187 persons, each person being examined



Maladjustments agreed upon by two examiners working independently on 35 cases of Diabetes Mellitus.

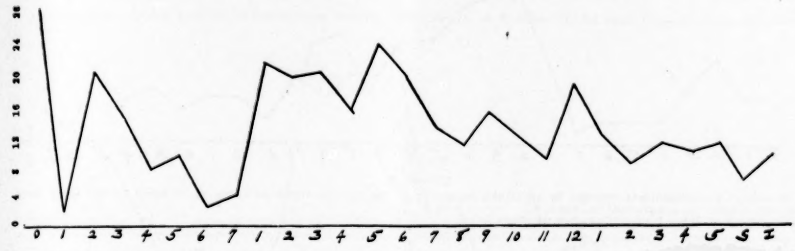


CHART 1.

Structural maladjustments recorded by all of the examiners at the various vertebral levels in 35 cases of Diabetes Mellitus as summarized in Table 1. The vertical column of figures to the left indicates the number of maladjustments recorded at various vertebral levels as charted. The figures under each chart indicate the various vertebrae of the cervical, dorsal and lumbar region. S = sacrum. I = ilium. The upper chart shows the number of maladjustments which were agreed upon at any given level in the same patient examined independently by two observers. The lower chart shows all maladjustments recorded by all of the observers.

DIABETES MELLITUS

	A & B	A	B	A & C	A	C	D & E	D	E	ABC DE	Two
	13	13	13	16	16	16	6	6	6	35	35
0	11	4	6	8	1	4	0	0	0	29	5
1	0	0	1	0	0	1	0	0	0	2	0
2	4	2	7	3	1	1	3	2	2	20	5
3	4	1	4	4	1	1	1	0	0	14	2
4	2	0	4	0	0	1	0	0	0	7	0
5	3	1	3	1	0	2	0	0	0	9	1
6	0	0	1	0	0	1	0	0	0	2	0
7	1	0	1	2	0	0	0	0	0	4	0
1	5	2	4	5	2	3	3	0	1	21	4
2	4	0	3	5	1	3	3	1	1	19	2
3	4	1	3	7	1	1	5	0	0	20	2
4	1	1	6	3	1	1	3	1	1	15	3
5	4	1	7	3	3	6	2	1	1	23	5
6	2	2	6	4	3	3	1	1	3	19	6
7	1	1	5	2	1	3	1	0	0	12	2
8	3	0	3	3	0	1	0	0	0	10	0
9	2	2	7	4	0	1	0	0	0	14	2
10	3	1	3	2	1	2	1	0	0	11	2
11	1	0	3	2	0	1	1	0	0	8	0
12	2	0	7	3	1	5	1	0	0	18	1
1	3	0	2	2	2	3	1	0	0	11	2
2	0	0	3	2	0	1	1	0	0	7	0
3	0	0	4	2	1	3	1	0	0	10	1
4	3	1	4	1	0	1	0	0	0	9	1
5	3	1	4	2	0	1	0	0	0	10	1
S	1	0	2	1	0	0	0	0	1	5	0
I	0	0	3	3	1	2	0	0	0	8	2
										337	49

TABLE 1

The left-hand column of figures represents the vertebrae of the various vertebral levels—cervical, dorsal and lumbar. S = sacrum, I = iliac. The columns headed A, B, C, D and E show the maladjustments recorded by each of the observers, while A & B, A & C, D & E show those maladjustments agreed upon by two observers on the same patient. The column ABCDE shows the sum of the maladjustments recorded by all of the men at the various vertebral levels, with those maladjustments agreed upon by any two observers shown in the last column. The letters A, B, C, D and E refer to each of the five observers in this work, while the numbers just under them indicate the numbers of patients examined in each case.

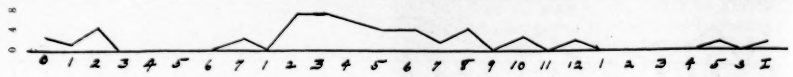
by two of five men who worked at various times on the problem. The examiners were made up of two practicing osteopaths, two men trained as regular physicians and one trained as an osteopath who was also in his last year of medical school during the time that he worked on this problem.

The cases have been grouped according to the diagnosis where there were a sufficient number of any one disease to be of value. These represent the diabetic, cardiac, and respiratory groups. All of the other types of cases examined have been grouped as miscellaneous in the analysis of maladjustments.

Tables 1, 2, 3, and 4 show the number of cases in each group examined and the number of structural maladjustments recorded at each vertebra by the various examiners (A, B, C, D or E). In certain instances two examiners (AB, AC or DC) recorded the same maladjustments in the same patient. These have been recorded in the extreme right hand column of the tables.

The figures in the left hand column of the tables represent the various vertebral levels, cervical, dorsal, lumbar, sacral and the iliac bones. The second column from the right in the tables is the sum of the maladjustments found by the five examiners.

A fact brought out very strikingly by all of the tables and even better by the graphs is that numerous maladjustments have been recorded at nearly every vertebral level; but the same



Maladjustments agreed upon by two examiners working independently on 25 cases having some form of cardiac disease.

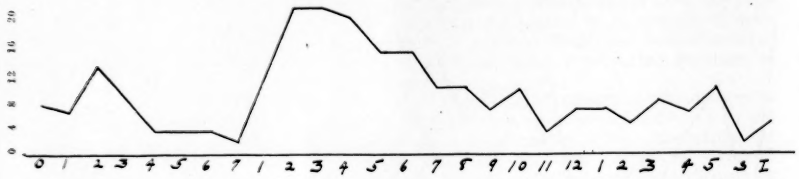


CHART 2.

Structural maladjustments recorded by all of the examiners at the various vertebral levels in 25 cases having some form of cardiac disease as summarized in Table 2.
See legend of Table 1 for explanation of chart.

CARDIAC

	A	A & B	B	D	D & E	E	ABC DE	Two
	4	4	4	21	21	21	25	25
0	2	1	2	2	1	1	7	2
1	2	1	2	2	0	0	6	1
2	0	0	1	7	3	4	12	3
3	0	0	2	4	0	2	8	0
4	0	0	2	1	0	9	3	0
5	0	0	2	1	0	0	3	0
6	1	0	0	1	0	1	3	0
7	0	0	0	1	1	1	2	1
1	0	0	1	4	0	6	11	0
2	3	1	2	10	4	5	20	5
3	2	1	2	11	4	5	20	5
4	0	0	3	10	4	6	19	4
5	0	0	2	8	3	4	14	3
6	1	0	0	9	3	4	14	3
7	1	0	2	5	1	1	9	1
8	2	1	1	4	2	2	9	3
9	2	0	0	3	0	1	6	0
10	2	1	2	3	1	2	9	2
11	0	0	1	1	0	1	3	0
12	2	0	0	3	1	1	6	1
1	1	0	2	3	0	0	6	0
2	0	0	0	3	0	1	4	0
3	1	0	0	5	0	1	7	0
4	1	0	1	4	0	0	6	0
5	3	0	1	4	1	1	9	1
S	0	0	0	1	0	0	1	0
I	2	1	1	1	0	0	4	1
							221	36

TABLE 2

See legend under Table 1.

lesion was recorded by two examiners independently in a very small number of instances. In the 187 cases a total of 1889 lesions was recorded with only 331 of these or 17.5% recorded by two observers at a given level. In many cases bony maladjustments were recorded by two observers at a given level which were exactly opposite malpositions. These have not been shown separately in any of the tables but of course would tend to reduce the actual percent of agreements. On the other hand many obese patients were examined in whom it was very difficult to count the vertebrae accurately. This would account for many failures to record the same structural maladjustments.

Chart No. 1 shows in graphic form the results of examination of 35 patients with diabetes mellitus as recorded in the two right hand columns of Table No. 1.

It will be seen that 337 maladjustments were recorded with only 49 or 14.5% recorded by two examiners. There were 14 maladjustments recorded by two observers at the same level which were diametrically opposite as to mal-position of the bony structure.

Chart No. 2 is the same for 25 patients affected with some form of cardiac disease recorded in Table No. 2.

221 maladjustments were recorded with 36 or 16.2% agreements and 4 recorded being diametrically opposite.

The graph of Chart No. 3 represents the two right hand columns of Table No. 3. 61 respiratory tract cases were examined with 568 maladjustments recorded, 119 or 20.9% the same by two observers and 12 opposite by two observers.

The striking thing about the graphs of the various diseases is the similarity of the curve in each. If structural maladjustment leads to perverted function (disease), the curve for a given perverted function should show a distinct prominence at the vertebral level said to control that particular function. The lesions are recorded at nearly every level with little variation at any point except between the first and sixth dorsal. This portion of the curve is prominent in all of the graphs shown. The prominence seems more marked in Chart 3 and might suggest that a more constant maladjustment was found in this area in the respiratory diseases than in the others. A study of Chart 4, in which the broken line represents the maladjustments in 61 respiratory cases while the continuous line represents the results of examination of the 66 miscellaneous cases of Table 4, will show the fallacy of this conclusion. It will be seen that the

Maladjustments agreed upon by two examiners working independently on 61 cases having some form of respiratory disease.

CHART 3.

Structural maladjustments recorded by all of the examiners at the various vertebral levels in 61 cases having some form of respiratory disease as summarized in Table 3.
See legend of Table 1 for explanation of chart.

	Respiratory											
	A &			A &			D &			ABC	
	A	B	B	A	C	C	D	E	E	DE	Two
	7	7	7	16	16	16	38	38	38	61	61
0	3	0	2	0	1	3	2	0	4	14	1
1	1	1	2	4	0	1	3	1	4	15	2
2	2	2	5	3	1	1	13	3	8	32	6
3	3	0	2	2	0	1	11	3	6	25	3
4	2	1	2	2	1	2	6	3	6	20	5
5	0	0	1	0	0	0	5	3	3	9	3
6	0	0	0	0	0	1	6	3	5	12	3
7	0	0	0	3	0	0	6	3	5	14	3
1	2	0	1	3	2	2	6	3	9	23	5
2	3	1	2	7	4	5	17	10	15	48	15
3	1	1	4	6	3	4	20	10	14	49	14
4	3	1	1	9	5	7	14	3	7	41	9
5	2	2	4	10	5	7	11	6	14	48	13
6	1	0	1	3	3	5	9	4	6	25	7
7	4	3	3	1	1	2	10	4	6	26	8
8	2	1	3	1	0	0	6	2	6	18	3
9	2	0	0	1	1	2	7	3	7	19	4
10	0	0	1	0	0	0	7	1	5	13	1
11	2	1	2	2	0	1	9	2	4	20	3
12	1	0	4	3	0	2	6	2	5	21	2
1	0	0	2	4	1	1	7	1	1	15	2
2	0	0	2	1	0	3	6	0	0	12	0
3	1	1	2	2	0	1	4	0	0	10	1
4	2	1	1	2	0	0	4	0	1	9	1
5	4	2	3	3	1	1	4	1	1	16	4
S	1	0	0	0	0	1	2	0	1	5	0
I	3	1	3	1	0	0	2	0	0	9	1
										568	119

TABLE 3

See legend under Table 1.

curves are practically parallel throughout in a series composed of approximately an equal number of cases. The ease of palpation of the upper dorsal spine may help to account for the large numbers of lesions recorded in this area although there may be other factors to consider.

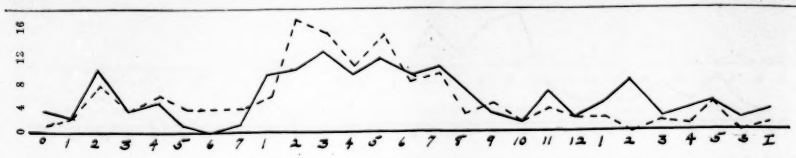
The miscellaneous cases are made up as to diagnosis as follows: Hypertension 14 cases, Pernicious anemia 5 cases, Nephritis 5 cases, Psychoneurosis 8 cases, various gastric and intestinal disorders 13 cases, and finally one or two cases each of several minor conditions including one or two diagnosed as having no disease.

CONCLUSIONS

Several rather obvious points concerning structural abnormality are brought out by the above charts and tables representing the result of the examination of the vertebral column of 187 patients in the wards of the Peter Bent Brigham Hopital by two of five men working at various times.

1. Muscle spasm and tenderness along the vertebral column was found in this group of cases to be too variable and unreliable to be recorded as a definite and separate structural maladjustment.

2. Structural maladjustments were found scattered indiscriminately along the vertebral column, especially in the upper half of the dor-



Maladjustments agreed upon by two examiners working independently on 66 miscellaneous cases are shown by the continuous line.

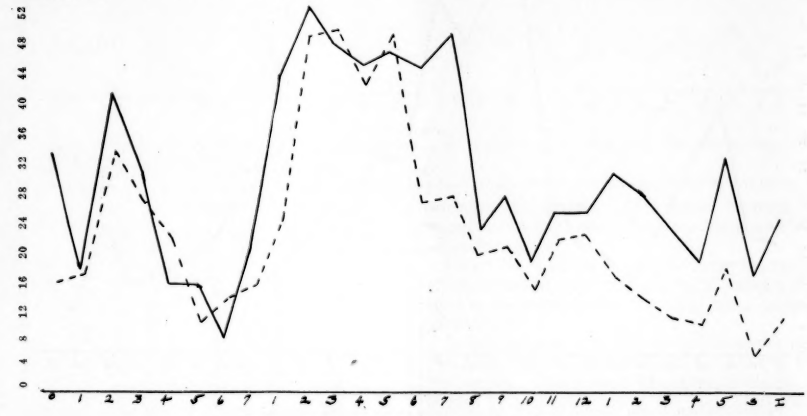


CHART 4.

Structural maladjustments recorded by all of the examiners at the various vertebral levels in 66 miscellaneous cases as summarized in Table 4 are shown by the continuous line.
The broken line represents the respiratory cases of Chart 3.
See legend of Table 1 for explanation of chart.

	MISCELLANEOUS											
	A &			A &			D &			ABC		
	A	B	B	A	C	C	D	E	E	DE		Two
	25	25	25	12	12	12	29	29	29	66		66
0	14	3	10	4	0	0	2	0	2	32		3
1	3	1	5	5	1	1	0	0	2	16		2
2	8	4	10	1	0	0	10	4	11	40		8
3	7	1	9	3	1	3	3	1	4	29		3
4	4	2	6	0	0	0	2	2	2	14		4
5	3	1	9	0	0	1	0	0	1	14		1
6	2	0	0	0	0	2	1	0	2	7		0
7	3	0	7	1	1	1	5	0	2	19		1
1	10	2	8	2	0	1	13	6	8	42		8
2	8	2	13	5	1	1	14	6	11	52		9
3	4	2	13	4	1	4	12	8	10	47		11
4	9	4	10	3	1	2	14	3	6	44		8
5	7	3	13	5	0	0	11	7	10	46		10
6	9	4	11	2	0	1	8	4	8	39		8
7	9	5	15	4	0	2	9	4	8	47		9
8	4	2	5	0	0	0	9	4	4	22		6
9	3	0	8	4	1	2	5	2	4	26		3
10	2	0	4	1	0	3	6	1	1	17		1
11	4	1	8	3	1	2	4	3	3	24		5
12	4	0	9	3	0	0	5	2	3	24		2
1	7	0	5	2	1	3	7	3	5	29		4
2	6	4	5	1	0	2	6	3	6	26		7
3	7	1	5	1	0	4	1	1	3	21		2
4	10	3	5	1	0	0	0	0	1	17		3
5	12	2	6	4	1	1	4	1	4	31		4
S	2	0	2	2	1	4	2	1	3	15		2
I	7	3	10	2	0	1	3	0	0	23		3
										763		127

TABLE 4

See legend under Table 1.

sal spine and regardless of the disease process from which the patient was suffering.

3. No predominant structural abnormality was found in any series of cases suffering from a given perversion of function (disease).

The authors wish to express their appreciation for advice received from Dr. Channing Frothingham and for help in examination of patients by Dr. Ross Golden, Dr. L. R. Whitaker and Dr. C. H. Downing.

Buffalo Studies Its Infant and Maternal Deaths

THE Buffalo Foundation has just completed a thorough study of infant and maternal mortality in that city. The study was conducted in coöperation with the City Registrar of Vital Statistics and was based upon an analysis of Department of Health records for 1922 and 1923. One of the interesting facts brought out in the study was the wide variation in the death rates for babies in the different wards of the city, four wards having rates under 60 per 1,000 live births, and three wards rates above 131. Knowing now where the problem is most serious, Buffalo plans to eliminate so far as possible the conditions which threaten the lives of her babies.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 11121

MEDICAL DEPARTMENT

An American weaver of thirty-nine entered December 31. Two of his brothers died of consumption. He had measles in childhood and frequent sick headaches in his youth. At twenty-five he had gonorrhea and at thirty-one typhoid fever. Since that illness he had never been strong and his headaches had been more severe. For eight years his eyes had been prominent. At thirty-six he had "malaria," —fever without chills for six weeks. He took a glass of whiskey a week and an occasional glass of beer.

Three months before admission he began to have severe constant generalized headache with some nausea. He vomited every other day and felt weak and exhausted. He gave up work and was in bed off and on for a day or two at a time. At the end of a month the headache left him, but the vomiting continued. He lost weight and strength. Four weeks before admission the vomiting became more severe. Since that time he had vomited nearly everything taken. His bowels were constipated, not moving for four days. He urinated three or four times at night. Two weeks before admission he began to have dyspnea, which had grown steadily worse. Six days before admission he began to have edema of the feet and legs which increased rapidly for three days, then subsided somewhat. For two weeks he had had considerable nosebleed every two or three days.

Examination showed a very round shouldered, fairly well nourished man looking haggard and sick. His skin was dark, his mucous membranes pale. The teeth were poor, many missing. The throat showed three small ecchymoses on the uvula and the posterior pharyngeal wall. The apex impulse of the heart was seen and felt in the fifth space 4½ inches to the left of the midsternum, half an inch outside the nipple line, coinciding with the left border of dullness. The right border of dullness was an inch and a quarter to the right of midsternum. The action was regular, the sounds rather loud, the aortic second sound sharp and ringing. A rather hissing early systolic murmur was heard slightly over the whole precordia, best at the apex, and transmitted a short distance into the axilla. The pulses were normal. The systolic blood pressure was 210. The lungs showed a few fine moist râles at both bases behind. The liver dullness extended to the iliac crest. An indistinct edge was felt. There was much tenderness over the liver. There was marked soft edema of the feet. The pupils reacted very slightly. The other reflexes were normal.

The temperature was 97° to 98.3° with a terminal rise to 102°. The pulse was 97 to 110. The respirations were 9 to 31. The output of urine was normal, the specific gravity 1.007-1.008. There was a slight trace of albumin at both of two examinations. The sediment showed pus at one. The hemoglobin was 55%, the leucocytes 8,600, the polynuclears 90%, the reds 2,972,000, with no marked achromia, poikilocytosis or polychromatophilia and no stippling.

The patient was unable to lie down, slept very poorly even with opiates, and grew rapidly worse. He vomited several times. The edema diminished. His respirations slowed down to six or eight a minute with no opiates. Unless roused he lay with his eyes rolled up and lids half closed. When roused, however, the respiration improved and he looked bright. The night of January 3 he became very delirious and sank rapidly. The morning of January 4 he quietly died.

DISCUSSION

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

In general people after typhoid fever are well. It is not like influenza, for instance, which often leaves people crippled. Typhoid patients usually are just as well as they were before,—sometimes fatter than before. It is well known that after typhoid fever people gain flesh, whether because they eat so much in convalescence or for some other reason I do not know.

That illness at thirty-six was probably not malaria, because in this part of the world we do not have fever of the malarial type without chills, though in the tropics we do.

At the end of this history things are fairly clear. It begins in a very obscure way but gets clearer as we go on. We have a longstanding headache. We have a longstanding nausea. We have nycturia, dyspnea, edema and nosebleed. This whole group of symptoms points in one direction and only one direction, and that is nephritis. That is the only disease which will cover all those symptoms. That does not explain his previous fever, but his previous fever may have been the cause of his nephritis. Nephritis often comes from infection. So that as we begin the physical examination I distinctly expect to find confirmation of the hypothesis of a chronic nephritis.

NOTES ON THE PHYSICAL EXAMINATION

These ecchymoses were presumably of the purpuric type which is often seen in such a disease as I am here expecting.

Everything in the physical examination backs up the guess made on the basis of the history. That is, he has an enlarged heart, a sharp aortic second, a high systolic blood pressure, with evidence of passive congestion of the liver and of the lungs.

I take it there were only two examinations of the urine, so we may suspect but we do not know that the specific gravity is fixed.

I do not know the explanation of the slow respiration without opiates.

DIFFERENTIAL DIAGNOSIS

He was here only five days. It is a perfectly clear case, so far as the record goes, of chronic nephritis, hypertrophy and dilatation of the heart, passive congestion of the organs, and death from uremia.

Just what we mean by "uremia" is a question not easily answered. Some people are willing to say now that by uremia we mean a high blood pressure, and that we never have real uremia without that. I do not think that is true. On the other hand the chemical basis for uremia is not so good as it used to be. We certainly get all the clinical signs of uremia in cases that do not have high retention products. I think we are all in the air at present. We cannot distinguish uremia, for instance, from the end stage of cirrhosis of the liver. In eclampsia (which sometimes has a normal kidney) we may get symptoms indistinguishable from uremia. There we have a high blood pressure ordinarily. That is one of the reasons why we tend to say that uremia means high blood pressure. But I do not think the matter is quite so simple as that. I am perfectly clear that in some cases the diagnosis of uremia has been made when post-mortem we found no disease of the kidney but did find a high blood pressure, and I have no doubt that a good many diagnoses of chronic nephritis are wrong because made on the basis of supposedly uremic symptoms due to high blood pressure.

A PHYSICIAN: What about the early hissing systolic murmur?

DR. CABOT: I do not believe it means anything wrong inside the heart. We almost always get it with a high blood pressure. What it is due to I have no idea.

DR. RICHARDSON: Was there any temperature?

DR. CABOT: There was a terminal temperature. Although the leucocytes were low in total count the polynuclears were ninety-eight. So that there is a good chance of some terminal infection. If I am asked what, I cannot answer. The commonest in chronic nephritis is acute pericarditis, the next is pneumonia. We have no evidence of either of those.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Chronic nephritis.
Uremia.
Myocarditis.
Arteriosclerosis.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic nephritis.
Uremia.
Hypertrophy and dilatation of the heart.
Chronic passive congestion.
Terminal infection.

ANATOMICAL DIAGNOSIS

Chronic interstitial nephritis.
Hypertrophy and dilatation of the heart.
Septicemia, streptococcus.
Pneumonia.
Soft hyperplastic spleen.
Foci of obsolete tuberculosis of the lungs.

DR. RICHARDSON: The head was not examined in this case. There was a slight amount of fluid in the peritoneal cavity. The liver at the time of necropsy was only three fingers down in the right mammillary line. It makes a difference where the line is that is taken when the liver is measured. The distance between the lower margin of the right lobe of the liver in the anterior axillary line and the crest of the ilium is pretty short. But if taken in the right mammillary line the liver might have been much farther up.

DR. CABOT: We have noticed over a long period of time a great many observations in which clinically we got a very big liver and post-mortem you got passive congestion. Are the anatomical conditions such that you can easily imagine a liver greatly engorged in life and collapsing after death?

DR. RICHARDSON: Yes, to a certain extent, but not so marked as in this case.

DR. CABOT: Have you ever tried the experiment of blowing up the liver to see if it can be made to reach this size?

DR. RICHARDSON: No.

DR. CABOT: It is always in this type of case that these discrepancies come. They do not come in leukemia, where we have a tough firm liver. It is always in the circulatory cases that we differ from the post-mortem findings.

DR. RICHARDSON: The diaphragm on the right was at the fifth rib, on the left at the fifth interspace. That is about right, although with these levels the pleural cavities were full of fluid,—hydrothorax. The pleura was coated with fibrinous exudate in places, and the lungs showed here and there small focal areas of frank pneumonia, otherwise some congestion.

There was moderate hydropericardium. The heart weighed 740 grams. That is a pretty large heart. The myocardium was pale, opaque. Some question was raised at the time about the myocardium. We made a histologi-

cal examination. There was no increase of interstitial tissue, but a note was made that the individual fibers seemed to be enlarged. The cavities were increased in size. The mitral valve measured twelve cm., the aortic eight, the tricuspid thirteen and a half. But there were no lesions of the valves, nothing within the heart to account for its size.

The aorta was fairly smooth; no definite amount of arteriosclerosis except that in the renal arteries there was some, not very marked. I mean by that, the artery that runs from the aorta to the kidney and the first branches where it breaks up into kidney substance. As a matter of fact we found later that there was considerable sclerosis of the arteries in the kidney substance.

The liver weighed 2190 grams. That is a large liver, but still only three fingers down. The tissue was slightly doughy and cinnamon brown in color.

The spleen weighed 240 grams, slightly soft, the follicles and trabeculae visible; some congestion and some softness from the infection present, a streptococcus septicemia.

The combined weight of the kidneys was 200 grams. That is 740 grams of heart against 200 grams of kidney. Even with small kidneys that is a markedly hypertrophied heart. The question from the anatomical standpoint at least is raised as to whether there may not have been some hypertension. The capsules came off fairly easily, leaving granular surfaces generally. The tissue was tough, the markings indistinct, the cortex varying from two to four mm., the cut ends of the vessels prominent. On the whole, macroscopically an arteriosclerotic nephritis, and the histological examination bore that out. There was no evidence of any glomerulonephritis.

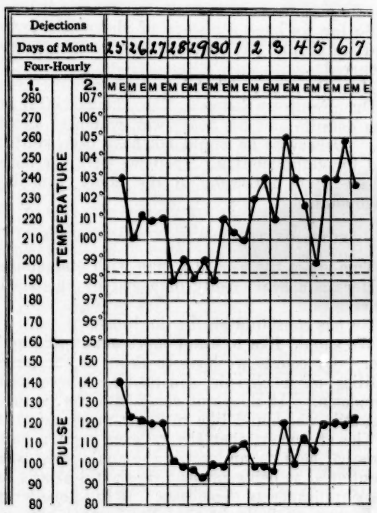
The gastro-intestinal tract showed a velvety mucosa, dull reddish, juicy,—passive congestion.

CASE 11122

Medical Department

An electrician of twenty-seven came to the Accident Room November 25. His mother died of a paralytic stroke. Except for an attack of typhoid fever seven years before admission and for rheumatic fever confining him to bed for three months he had been well. He drank two or three glasses of beer daily and an occasional glass of whiskey. He smoked thirty cigars, six boxes of cigarettes and forty cents worth of plug tobacco weekly. His present illness began four days before admission with sharp pain in the side, chill, and cough, all of which had persisted, with considerable fever. His appetite and sleep were poor.

Examination showed a well nourished man with pallor of the skin and mucous membranes. The apex impulse of the heart and the left border of dullness were in the fifth space just outside the nipple line, 4½ inches from midsternum. There was no enlargement to the right. The sounds and action were normal. The first sound at the apex was markedly accentuated, heard over the whole precordia. A presystolic and a systolic murmur were heard at the mitral area, loudest at the apex, transmitted to the axilla. A soft diastolic murmur was heard at the aortic area. The pulmonic second sound was greater than the aortic. The pulses and arteries were normal. The right upper chest in front and back showed dullness, increased vocal and tactile fremitus and whispered voice; the front showed also bronchial breathing. At the right base behind were many fine moist râles. The abdomen showed slight general tenderness. There was a nodule in the right epididymis. The knee-jerks were not obtained. The other reflexes were normal. The temperature and pulse were as shown in the chart.



The respirations were 28 to 45. The urinary output was 65 to 85 ounces until the day before death, when it fell to normal. The specific gravity was 1.020 to 1.032. There was a trace to a slight trace of albumin at two of three examinations and a few to rare red blood corpuscles at two; no sugar. The hemoglobin was 100 per cent., the leucocytes 11,000 to 20,000.

November 29 the temperature had dropped to normal. Coarse moist râles were heard over the right back and at the upper right front. December 2 a diastolic murmur was heard best in the third left interspace, but also in the second right interspace. Fine moist râles were heard all over the base of the right lung behind and at the side. The patient complained of pain in the

right shoulder joint and of sharp pain in the splenic region. December 7 the diastolic murmur had become very loud and could be heard all over the precordia. The heart sounds were very loud and snapping. The pulse at the wrist was of rather poor volume and tension. The patient had failed rapidly during the past two days. That night the breathing was somewhat harsh at the left apex and in the axilla. Vocal and tactile fremitus were somewhat increased. The evening of December 8 he suddenly took a turn for the worse, sank rapidly, and in three hours died.

Discussion

BY DR. MAURICE FREMONT-SMITH

NOTES ON THE HISTORY

Of course we should like to know at what age his mother died. Otherwise this history does not help us.

So far we have a history of rheumatic fever, and we should like to know how long ago that occurred.

Miss Painter: That is not given.

Dr. Fremont-Smith: The rheumatic fever was definite and his heart was evidently affected. Otherwise he would not have been kept in bed for eight months. Then we have the onset of an acute infection in the chest, probably pneumonia.

NOTES ON THE PHYSICAL EXAMINATION

This is not the look of a man with pneumonia.

Here is a heart badly damaged by rheumatic fever.

He has consolidation in the lungs.

We should like to know more about this nodule. Of course we have to think of the possibilities of a chronic prostatitis, tuberculosis, neoplasm. It does not come into the picture, but still we should like to know more about it.

The statement that the reflexes were normal does not help us, and yet if the ankle-jerks were mentioned as being present we should attach little significance to the absence of the knee-jerks.

I very much doubt that the hemoglobin was one hundred per cent.

DIFFERENTIAL DIAGNOSIS

It is quite evident from the story that there was an acute endocarditis superimposed upon an old rheumatic infection. The question comes up whether we may explain the whole picture as the result of a bacterial endocarditis involving the valves injured previously by the rheumatic fever and resulting in infarction. Of course in rheumatic fever the left side of the heart is usually involved. However, the tricuspid is very frequently involved, although we are rarely able to make a diagnosis during life. It would be possible to assume a subacute bacterial endocarditis with vegetations in which the first symptom might be an infarct from the tricuspid valve, infarct into the lung with pain, cough and temperature.

The sharp pain in his spleen, the few red cells in the urine, all fit in the picture of multiple emboli. The usual history of pulmonary infarct is death from infarct, that is, if the man died after having had several small pulmonary infarcts we should expect a death from infarct, and the description of his death here is not that of infarct.

We have of course to think of the perfectly good possibility that we have here a bronchial pneumonia, that the infection on the heart valves was secondary to that, a secondary streptococcus acute endocarditis, and that we have here a recovery of one part of the lung with the later involvement of another lobe. I hope that we may be able to explain all on the basis of a subacute or acute bacterial endocarditis with multiple infarcts to the lungs, kidneys, spleen.

Dr. Cabot: Have you anything to say as to that pain in the right shoulder joint?

Dr. Fremont-Smith: Nothing except that infarcts are common to the tissues about the joints. One gets frequently I believe pain, usually in the smaller joints around the fingers.

Dr. Cabot: I was thinking of the way we see hepatic pain sometimes referred to the shoulder joint, pain believed to originate in the gall-bladder, and wondering whether we could make a liver embolus out of the symptom in this case.

Dr. Fremont-Smith: With diaphragmatic irritation.

Dr. Cabot: Yes. It is just a guess.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Croupous pneumonia.
Arthritis.
Mitral and aortic disease.
Acute endocarditis?

DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Chronic and acute endocarditis.
Multiple infarcts to the lungs, kidneys and spleen.
Possibly bronchial pneumonia.

ANATOMICAL DIAGNOSIS

Pneumococcus septicemia.
Lobar pneumonia.
Organizing pneumonia.
Chronic and acute endocarditis of the mitral, aortic, tricuspid and pulmonary valves.
Hypertrophy and dilatation of the heart.
Infarcts of the spleen and kidneys.

Dr. Richardson: We were not permitted to examine the head.

The lungs were voluminous. The pleura here and there was coated with fibrinous exudate. Of course there was some passive congestion in the lungs, and in addition in the up-

per half of each lung areas of pneumonia which in portions resembled a pneumonia in the stage of resolution and in places an organizing pneumonia,—that is, an older combined with a more recent pneumonia.

The heart weighed 493 grams,—considerably hypertrophied—and all of the valves, the mitral, aortic, tricuspid and pulmonary, showed chronic endocarditis in the form of fibrous deforming thickening with if anything decrease in their circumferences,* and on this basis what is called here polypous endocarditis, that is, smaller and larger soft frank masses of vegetations. That is an acute endocarditis on top of an old. The organism is the pneumococcus all through.

A pneumococcus septicemia, and in the vegetations on the heart valves many pneumococci.

The kidneys were not remarkable except for infarcts. There was a hypertrophied spleen, in places soft, and with infarcts here and there.

I could not make out any infarcts of the lungs but of course with a source for emboli as there was on the tricuspid valve there might have been some small ones, but none were found.

DR. FREMONT-SMITH: I think we have to assume that the pneumonia was primary, don't we?

DR. RICHARDSON: Yes.

DR. CABOT: That is, you believe that the heart trouble came from the pneumonia?

DR. RICHARDSON: Yes, the acute trouble.

DR. FREMONT-SMITH: Do we ever get a primary pneumococcus septicemia without a pneumonia?

DR. RICHARDSON: Yes.

DR. G. C. CANER: Wouldn't you expect a more sudden onset if it were? There is a long history of smoking up to his admission.

DR. CABOT: Yes. That is a good point. People with acute endocarditis do not smoke that way. Isn't it true, on the other hand, that these vegetations are bigger than those you ordinarily see in a heart accompanying pneumonia? Aren't the vegetations accompanying pneumonia generally little ones?

DR. RICHARDSON: I don't think one can make a definite statement in regard to that.

DR. FREMONT-SMITH: Is pulmonary infarct a common symptom in subacute bacterial endocarditis? There is no reason why it should not occur, but does it happen?

DR. CABOT: I do not remember it. I do not believe it is common.

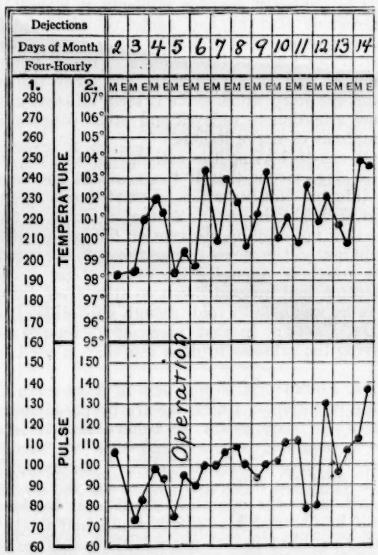
*The mitral valve measured 8 cm., the aortic 7 cm., the tricuspid 8½ cm., the pulmonary 7 cm.

CASE 11123

SURGICAL DEPARTMENT

An Irish stonecutter of fifty-eight entered September 2 for relief of difficulty in urination of two or three years' duration. He had always had the best of health, though he had lost considerable weight, he did not know how much. He denied venereal disease. Five days before admission he had complete retention. A physician catheterized him and left the catheter in for two or three days. Since the morning before admission he had passed some urine with difficulty. He voided last four hours before admission. Since the onset of the acute retention he had had continued pain in the bladder.

Examination showed a man looking sick and old and showing evidence of the loss of considerable weight. His color was fair, his mucous membranes rather cyanotic. The location of the apex impulse of the heart is not recorded. There was no enlargement to percussion. The radials were slightly hard. There was moderate lateral excursion of the brachials. The action was regular, the sounds faint but of fair quality. There were no murmurs. The prostate was slightly nodular but not enlarged or tender. The examination was otherwise negative. The temperature and pulse are shown in the chart. The respiration was normal until September 7, afterwards 24 to 30 with a rise to 40 on the last two days. The urine was normal in amount, cloudy, alkaline at one of two examinations, the specific gravity 1.006-1.018, a slight trace of albumin at the second examination. The blood is not recorded.



The patient was put immediately upon constant drainage. There was a residual of four ounces of slightly turbid urine. He complained of considerable pain in the perineum. The catheter was removed and the perineum found

to be fluctuant and tender about the bladder. Pressure expressed pus from the meatus.

Operation was done September 5. The patient did not gain as he should have done after it. The urine was kept up by rectal salt solution. September 13 he was worse and markedly jaundiced. There was bile in the urine and in the stools. The amount of urine was greatly diminished. During the last few days of his life a tremendous systolic blow was heard principally over the apex beat. September 14 he died.

Discussion

by dr. edward l. young, jr.

On the story alone it is pretty certain that this man had an obstructing prostate, because a local physician had catheterized him and a retention from stricture would not have been catheterizable without some comment on the stricture. We know also that except in very rare cases stricture and prostate do not go together.

It is perfectly possible although it is highly improbable that he had acute retention from nerve lesion. Occasionally that is the man-trap which leads us into trouble. But it is not common, and as a rule there are other things about the story which make it seem unusual and draw attention to reflexes and other diagnostic symptoms of nerve lesions.

Of course leaving the catheter in for two or three days means that if the man did not have an infected bladder when he started this retention he did at the end. So that we have to figure now not only on obstruction but on cystitis. Apparently the acute congestion of the prostate which is generally the cause of the acute retention had quieted down so that after three days he was able to void, though with difficulty.

Dr. Cabot: Is that piece of technique of leaving the catheter in done less than it used to be?

Dr. Young: It is done less in these cases of acute retention. Unless we are prepared to go the whole way, unless we believe that it is a case demanding surgery, and then we should start at once giving drainage. Assuming a case which has to be operated on has not an infected urine the operation should not be done until he has had his infection and had it long enough so that he has got a little resistance to it. In the old days before we appreciated that I am sure some cases died because an immediate prostatectomy was done on the basis, "Here is a man in good condition, let us take his prostate out before anything hits him." The infection came when he was least able to handle it and finished him. So always that infection ought to take place in the course of drainage so that the patient gets resistance.

They give us the suggestion in the record that they think the prostate is not malignant. Of course the fact that it was not enlarged by rectum does not prove that there is not an intravesicular enlargement sufficient to cause obstruction.

There is just one thing that I should like to add, and that is the rather short history. It always makes us wonder whether or not there is a malignancy in the background, and here, with the loss of weight which he has had, it brings that suspicion a little more to the foreground.

In other words there was infection probably from the trauma of the original catheterization. I think the most important thing to remember about catheterization in all these cases is that gentleness is infinitely the most important thing. If I had to choose between two people one of whom would use all the asepsis in the world but who would not take particular care of the gentleness with which he worked, and another physician who spit on his catheter to lubricate it but was gentle, I would personally take the latter one every time, because the danger comes in the trauma and not in the number of bugs which happen to be roosting on the catheter when it is passed. The urethra contains bacteria anyway. This was in 1909, just about the time when we were changing a mortality in prostatectomy which was away up, fifty, sixty, seventy, eighty per cent., to the mortality which ought to exist today even in hospital cases of not over twenty per cent. Of course in the hands of men who do a great number of prostatectomies the mortality gets down as low as five to ten per cent.

Based on what we should say today, this man ought to have suprapubic drainage to get rid of the infection, to give the kidneys good drainage, to find out what is there. As a matter of fact he was operated on three days after he came in, which today we should say is an absolutely inadequate amount of drainage, especially for such a case as this, unless in fact they merely opened the abscess.

On the backward look there is this other thing to remember, that a septicemia can occur from merely passing an instrument into the bladder. Two cases at least that I know of died of streptococcus septicemia after the passage of a sound as a routine. We know that this man has been infected both because of the infection of the urine, which is inevitable, and the periurethral abscess, so that he may be so badly septic that nothing will make any difference.

The operation may have been just drainage of the abscess or they may have gone right ahead and taken the prostate out at that time.

dr. young's pre-operative diagnosis

Obstructing prostate.
Cystitis.

operation

No pre-operative diagnosis is recorded. Gas and ether. Lithotomy position. A sound met obstruction in the deep urethra. One and a

half inch linear incision in the perineum with evacuation of two ounces of thick cloudy urine. The urethral bulb was surrounded by slough. A rubber catheter was passed through the perineum to the bladder. The wound was packed with iodoform gauze.

FURTHER DISCUSSION

In other words, a gangrenous periurethritis. The jaundice is a suggestion of sepsis which has hit the liver.

I assume that the systolic blow again was evidence of infection.

I do not think we can say anything more than that he died of sepsis started by urethral instrumentation which was done to relieve him of acute retention which it seems to me was probably due to congestive prostatitis. I do not see that we have any right to say that there was any nerve lesion, because we have no examination suggestive of it. I think Dr. Richardson will tell us there is a septicemia and the local evidence of trouble with slight enlargement of the prostate.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Perineal abscess.
Malignant endocarditis.

DR. EDWARD L. YOUNG'S DIAGNOSIS

Obstructing prostate.
Periurethral abscess.
Septicemia.
Acute endocarditis.

ANATOMICAL DIAGNOSIS

Malignant endocarditis of the mitral valve.
Septicemia, streptococcus.
Infarcts of the spleen and kidneys.
Icterus.
Operation wound.

DR. RICHARDSON: The incision in this case was a short one in the anterior abdominal wall. We could not remove any of the organs. Icterus was well marked. There was no evidence of peritonitis. There were no stones in the gallbladder. The hepatic, cystic and common bileducts were free, the mucosa negative; the bile flowed freely. The pancreas was not remarkable. The duct of Wirsung was free. There was no evidence of new growth in any of the organs seen, no ulcers in the lower end of the ileum. There was a soft hyperplastic spleen with infarcts. The kidneys were of normal contour and size and showed infarcts. The heart was somewhat enlarged and the mitral valve presented a frank mass of vegetations, three by two cm. The lungs were negative as nearly as could be made out.

The growth was streptococcus from the spleen. The vegetations on the mitral valve and endocardium showed the usual hyalin material, blood plates and masses of micrococci.

DR. YOUNG: Did you open the bladder?

DR. RICHARDSON: No.

DR. CABOT: As you hear the history, Dr. Richardson, would your guess be that this endocarditis originated in the conditions of the bladder and the instrumentation? He had had no heart symptoms, had he?

DR. YOUNG: There were none according to the record. They report the location of the apex impulse not recorded, no enlargement to percussion, the action regular, the sounds faint but of fair quality, no murmurs.

DR. RICHARDSON: When was the first rise in temperature?

DR. YOUNG: He came in with enough sepsis so that he had a temperature the day after he came in. The first rise was right after the operation. I believe he came in with a septicemia.

DR. RICHARDSON: But it shoots up on the day after operation. I think that is fair enough, but there is also the other side, that he may have had the septic condition mentioned and that following the operation it extended and set up the endocarditis.

CURRENT LITERATURE

ABSTRACTORS

GERARDO M. BALBONI — TRACY MALLORY
WILLIAM B. BREED — HERMAN A. OSGOOD
LAURENCE D. CHAPIN — FRANCIS W. PALFREY
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TUBERCULOSIS OF THE LARYNX

THOMSON, St. C. (*British Medical Journal*, Nov. 8, 1924) discusses at length tuberculosis of the larynx. He lays particular stress on the value of voice rest which can be so easily carried out in a sanitorium, but is elsewhere so difficult. Out of 119 cases which were cured, the healing was obtained without other local treatment than voice rest (whispers or silence) in no fewer than 73 instances. He finds that a laryngeal lesion generally indicates an extensive lung lesion, and has abandoned all local surgical methods of cure. The galvano-cautery he has never found to be harmful in a single case, although in some it has been abandoned as useless. It effected a cure in 46 out of 74 cases in which it was tried. Of these 46 cures, 29 are alive and well. Galvano-cautery is the best local treatment we have at present. In suitable cases it has replaced all other methods.

[R. C.]

SURGICAL TREATMENT OF MITRAL STENOSIS

CUTLER, E. C. et als. (*Archives of Surgery*, Part 2, Nov., 1924).

These authors present an article of 133 pages on this subject. This discusses the rationale for oper-

ative intervention, the diagnosis, and selection of cases for possible surgical treatment. They give a brief historical sketch, experimental data by the old methods, and experimental data with the new methods principally dependent upon a new instrument, the cardiovalvulotome. A description of operations with this instrument is given and discussion of the various surgical methods, their applicability to the treatment of mitral stenosis in man. They then discuss the operative procedure in four human cases. A brief summary, and an extensive bibliography is appended.

[E. H. R.]

Thrombo-Angitis Obliterans

Telford, E. D. and Stopford, J. S. B. (*British Medical Journal*, Dec. 6, 1924), take up the subject of thrombo-angitis obliterans in a most interesting article. They point out that "It is a disease of the larger blood vessels which, although of unknown origin, shows the histological features of an inflammatory lesion. The vessels mainly affected are the larger arteries of the limbs, but the disease is seen also to some extent in the veins. Thrombosis occurs and is followed by organization of the clot. This produces grave embarrassment of the circulation, alleviated to a variable extent by the usual canalization of the fibrous tissue." They cite 4 very interesting cases, and point out that the most important factor in treatment is the protection from any form of injury. It is for this reason very essential that a correct diagnosis be made early in the history of these cases. Where gangrene eventually ensues they think that it is important that nothing less than a supracondylar amputation of the thigh be done.

[R. C.]

Functional Mental Disorder Due to Toxaemia from the Bowel

Walker, J. (*The Lancet*, Nov. 22, 1924) reports 3 cases in which toxaemia from the bowel was responsible in producing symptoms and signs of disease affecting particularly the nervous system. They draw the following deductions from these cases:

1. The nervous system is susceptible to the influence of toxaemia either of exogenous or endogenous origin.
2. Toxaemia of intestinal origin is more frequent than is generally accepted, its deleterious action being more easily produced and more marked when the general health is either reduced by previous illness or the powers of resistance impaired by such agencies as heredity, personal neglect and malnutrition, and unhealthy environment.
3. Provided the toxaemia has not produced definite organic changes, it is quite safe to give a good prognosis. When recovery is complete an attempt should be made to explain to the patient how the illness was caused, in order to ensure a more satisfactory mode of living and at the same time considerably lessens the chances of recurrence.

[R. C.]

Conjugal Tuberculosis

Rowland, S. (*The Lancet*, Dec. 13, 1924) takes up the subject of conjugal tuberculosis. He summarizes his remarks as follows:

1. Conjugal tuberculosis is rare.
2. The death-rate from phthisis of the spouses of tuberculous persons varies little from that for the general population of a corresponding age.
3. Ideal conditions for infection exist, but infection rarely occurs owing to the age of greatest susceptibility having passed by the time the marriage age is reached.
4. My findings support the view that infection usually occurs during childhood, hence the advisability of protecting children whose parents are suffering from open phthisis. Upon these lines the Grancher system is founded, and is being worked successfully in France.
5. When conjugal infection does occur the disease usually runs a relatively short course.

[R. C.]

On the Treatment of Anaemia

Porteous, H. B. (*Edinburgh Medical Journal*, Nov. 1924) reports that "The injection of freshly prepared saline extracts of red bone marrow into the ear veins of rabbits caused in each case an immediate rise in the number of circulating erythrocytes; this rise was maintained during the period of administration of the extract, but on its cessation the count fell to normal in about two days." He found also that "similar results were obtained when normal humans were made the subjects of like experiments, the combined extracts being administered in the dried form in capsules containing 0.3 grams." He reports four cases, in the first of which satisfactory results were obtained, while in the last two unsatisfactory or negative results were obtained. He states that "a further series of tests seem to be indicated before any very definite conclusions can be drawn as to what type of case will derive most benefit from the administration of spleen and red bone-marrow extract."

[R. C.]

Xanthoma and Xanthosis

Stewart, M. J. (*British Medical Journal*, Nov. 15, 1924) takes up a number of different pathological states in which the deposition of cholesterin fat is the salient anatomical feature, and shows at the same time how frequent is the occurrence of minor grades of this condition in relation to a large series of pathological processes. He divides these lesions into groups as follows: In the first group are placed those in which the phenomenon is regarded as the local expression of a condition of hypercholesterinaemia—namely, cutaneous xanthoma, strawberry gall bladder, cholesteatoma of the choroid plexus, and arterial atheroma. The second group includes a long list of local lesions, necrotic, inflammatory, and neoplastic, in which the xanthosis is the result of absorption from disintegrating tissues or exudates.

[R. C.]

Wet Winds and Early Phthisis

Gordon, W. (*British Medical Journal*, Nov. 29, 1924) discusses, at length the relation between wet winds and early phthisis. He includes in his article numerous tables of interesting statistics, and concludes that the chance of becoming phthisical is at least twice as great in exposure to these winds as it is in shelter from them. He also finds that the chance of recovery is at least twice as great in shelter from these winds as it is in exposure to them, and that therefore there is no detail of treatment more vitally important to the patient than the placing of him in effective shelter.

[R. C.]

Lumbo-Sacral Backache

Verrall, P. J. (*British Medical Journal*, Nov. 1, 1924) emphasizes the following points: "In examining a case of lumbo-sacral backache first exclude gross disease, injury, and arthritis, and have a skiagram taken. Then look for outside mechanical causes and outside causes of overstrain of the nervous system. Finally, remember the paramount importance of the sympathetic system and seek for referred pain."

[R. C.]

Facial Irritability

M'Neil, C. (*Edinburgh Medical Journal*, Dec., 1925) in a most excellent article emphasizes the fact that there is a distinct clinical significance in facial irritability, and concludes as follows:

1. The clinical significance of facial irritability is very different in infancy (up to 2½ years), and in later childhood.

INFANCY

2. In infancy it is definitely associated with the convulsive disorders known as spasmophilia and including laryngismus, general convulsions, and tetany.

3. At this period of life, facial irritability indicates that spasmophilic convulsions (one or more kinds) have recently occurred, or may occur in the future.

4. The likelihood of return of active spasmophilia may be judged by the severity of the facial reaction, and by the degree of muscular atony present.

5. In the great majority of cases of spasmophilia bony rickets is also present.

6. The severity of the bony lesions of rickets is an unreliable guide to the danger of active spasmophilia.

7. In one-half of 56 cases of bony rickets, facial irritability was absent.

LATER CHILDHOOD

8. Over the age of 2½ years there is no apparent association of facial irritability with general convulsions, or with asthma. The great majority of cases of general convulsions at this period occurs without increased irritability of the peripheral nerves to mechanical stimulation.

9. At this period, facial irritability does not indicate that general convulsions have occurred or are likely to occur.

10. Facial irritability does not indicate a neuropathic constitution, or any recognised functional disorder or organic disease of the nervous system.

11. In most cases of older children, the sign has no definite nor serious pathological significance.

12. It is met with most frequently in cases of disordered digestion, mild or severe.

13. Its frequent occurrence in cases of coeliac disease suggests that it may be the result of disordered calcium metabolism in that disease, and in other conditions of chronic dyspepsia.

14. Its still more frequent occurrence in cases of rickets complicated with spasmophilia may be due to the same condition of disordered calcium metabolism.

[R. C.]

The Malarial Treatment of Paretic Neurosyphilis

Nolan D. C. Lewis, Lois D. Hubbard and Edna G. Dyar (*American Journal of Psychiatry*, Oct., 1924) in a long paper from St. Elizabeth's Hospital, Washington, D. C., review in detail the types of treatment used in general paresis, especially the artificial production of fever (malaria) as advocated by Wagner-Jauregg and others. Part II of this important paper is a statistical survey of the paretic admissions at St. Elizabeth's Hospital since 1886. Records of 1558 cases showed the average age of admission to be between 35 and 39, that over three-quarters of the patients died while in the hospital, mostly before the third year of hospitalization, and that only 9 cases "recovered." The hospital has always actively treated paretic patients and "of late years the salvarsan methods have been thoroughly tried and have served in the routine capacity. However, it cannot be said that the results have been particularly gratifying."

Sixty-eight paretic patients were selected for malarial treatment. Tertian malaria was used. In 31% "complete remissions" occurred, mental, physical, and serological, after several months of treatment. In the opinion of the authors, "this result would seem to justify extensive experimentation with the method."

Part III deals with histopathological studies of brains from four patients who died under malaria treatment. The usual signs of brain atrophy and gliosis were found, but the infiltration and exudate was decreased. Tendency to capillary hemorrhage and thrombosis was increased. In two brains no spirochetes were found, but in the other two, a few apparently damaged ones were demonstrated.

The paper contains a long list of the important references and is illustrated by ten plates.

[H. R. V.]

A Simple Method of Transfusing Pure Blood

Becart, A. and Churchill, A. (*The Practitioner*, Dec., 1924) after a study of 200 cases, are of the opinion that

1. In equal doses the transfusion of citrated blood gives rise to a more marked reaction than that of pure blood. This may possibly be due to a modification of the colloidal equilibrium of the plasma by the citrate solution.

2. The injection of small doses of pure blood—150 to 200 c.cm.—is practically free from any reaction.

They have devised a very simple method for blood transfusion by means of a special syringe by which blood can be withdrawn and injected under perfectly air-tight conditions. The advantages of this method are:

1. Pure, unmodified blood is used. No anticoagulant, no foreign substance is introduced into the recipient's circulation.

2. The transfused blood is brought into contact with a minimum of foreign matter. There are no taps, no rubber tubes, no grooves where the blood might stagnate and become coagulated. It is always in contact with paraffin.

3. It can be performed in any place.

4. No assistance is necessary.

[R. C.]

Solitary Serous Cysts of the Kidney

McKim, G. F. and Parke G. Smith (*Jour. of Urology*, Dec., 1924).

Solitary serous cyst of the kidney is a clinical entity and is one that should be regarded as a comparatively rare condition. Etiologically, it is dependent upon a congenital defect in development in the extreme portion of the renal cortex. Its symptomatology, while vague, is easily explained by the mechanical effects of the presence of a benign tumor mass. With the assistance of modern urological and radiological methods, its preöperative diagnosis should become more frequent. Reaction may be the more conservative procedure, but at the same time it may be the most hazardous to the patient.

[B. D. W.]

Surgical Horseshoe Kidney

Hess, Elmer (*Jour. of Urology*, Dec., 1924).

A favorable place for the obstruction to the ureter by a descending stone in a horseshoe kidney is at the point where the ureter crosses the connecting bridge, and the ureter usually crosses anteriorly. The good half of the kidney can be rendered useless either as a reflex phenomenon or by reason of its really being a direct part of the pathological half. While the author is not ready to make any general conclusions, in this case his severing the connecting bridge by the cautery without suture did not mean a urinary fistula. The good half, immediately the burden was taken away by the surgical removal, assumed its duties satisfactorily. The preöperative diagnosis of a horseshoe kidney is very difficult even under very favorable conditions.

[B. D. W.]

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DIETETICS AND THE VITAMINES

DIETETICS, or that branch of therapeutics dealing with food and drink in relation to health and disease, has assumed at the present time the importance which has long been its due. We understand now as never before that a certain diet makes for health and another diet for ill health and while our knowledge of dietetics as a science is by no means exact but still largely empirical, we are progressing by slow but sure stages to a good working understanding of the diet question. During recent years the matter of diet has been subjected to keen analysis by able medical men and chemists in various countries. Perhaps America has been especially conspicuous in this direction and as a result a satisfactory diet table can be drawn up for those in health and disease. From the preventive aspect diet bulks most largely, as it seems to be the fact undoubtedly that disease can be prevented frequently by a judicious selection of food eaten. The most intriguing investigations with regard to diet have been those which have been concerned with the vitamines, and in the line of research Americans have taken a leading part. It is only within the past twelve years or so that it has been found out that although a diet may fulfill all requirements according to the then recognized standards it may be still lacking in certain elements essential to the maintenance of life and nutrition. Of course these so-called accessory food factors are the vitamines, four of which are known in spite of the fact that their chemical composition has not been determined. A certain amount of haziness exists with respect to the vitamines and they have been referred to more than once as hypothetical.

A special number of the *Practitioner* published in January in which these substances are discussed by authorities at some length comes opportunely. This number of the *Practitioner* is headed "Dietetics: Vitamines and Food Reform," the title of the first paper contributed by Sir William Wilcox. It may be mentioned en passant that the *Practitioner* is a pioneer of numbers devoted to special subjects. The January issue of the *Practitioner* is the first of any prominent medical journal to be given over solely to dietetics and it may be added that no better collection of opinions on the matter has been presented to medical readers. Its contents, indeed, provide an embarrassment of riches from which it will be possible only to gather a few of the most outstanding points. Professor E. Gowland Hopkins, the Professor of Biochemistry in the University of Cambridge, and who has made the subject of vitamines almost his own and to which he refers as those elusive constituents of a complete dietary, clears up some moot points in his article. Professor Hopkins asserts that even though a young growing animal may consume a right proportion of the right kind of proteins and a well balanced supply of minerals, it may yet cease to grow and after displaying characteristic symptoms of malnutrition may quickly die; that is, if the food is wholly freed from vitamines. These elements of food have been partially isolated and are constituents of natural foods characterized by the fact that they exert a potent and indispensable influence upon metabolism, though present in strikingly small quantities.

Professor R. H. A. Plimmer, Professor of Chemistry in the University of London, reviews the vitamin problem in nutrition and in doing so points out a very important fact, and one that is overlooked frequently, that the highly refined and sophisticated foods of the day are sadly lacking in vitamines. He draws attention also to some extremely interesting facts, that during the war the slow healing of wounds was found to be associated with shortage of vitamin C. Heart and digestive troubles are seen with shortage of vitamin B and under all variations of vitamin shortage experimental animals are more susceptible to infections of all kinds. Professor Plimmer recommends strongly that the vitamin question shall be first attended to in the bringing up of infants and young children. In this way the foundation will be laid for a sound constitution, and, he adds, with our present day habit of eating refined and preserved foods attention to our vitamin supply should never be relaxed at any age.

Dr. John Boyd Orr, Director of the Rowell Research Institute, Aberdeen, lays most stress on the necessity for the mineral metabolism of the body to be well balanced. He is stated to have said once that if the mineral elements of a diet are ingested in due proportion that the vitamins will take care of themselves, that is, that the vitamines coëxist, so to speak, with the mineral elements. He also insists on the need for iodine in a diet. Professor Orr points out that the results of some recent work by Kelly at the Rowell Institute indicate that the addition of traces of iodine as little as 0.005 grams per day to a diet of cereal products fed to a growing hog increases the absorption and retention of calcium, phosphorus and nitrogen. It seems probable, therefore, that faulty assimilation, with resulting malnutrition, may occur through lack of iodine in diets consisting of cereals and other foods which have been subjected to processes in which whatever iodine may have been present originally is liable to be lost. Acting on this assumption potassium iodide has been added to the diet or the drinking water in certain districts and marked beneficial effects have been claimed. In this connection it may be pointed out that recently there have been numerous instances in America in which, in districts lacking in or free from iodine, goiter and other thyroid affections have occurred with considerable frequency. Professor Orr also draws attention to the fact that defective mineral metabolism is associated with a decreased, or probably decreased, resistance to infectious diseases. The concluding paragraph of Professor Orr's article is especially worthy of notice. "It is interesting that those foodstuffs which the results of modern research suggest as being of special value for the maintenance of health are those which have been recommended by experienced physicians for many generations."

Dr. S. Henning Belfrage of London gives an excellent article on the general and practical considerations of dietetics. Dr. Leonard Williams of London gives his views on present day food and manner of eating and cooking it. Dr. N. Mutch of London deals with diet as a mode of causing toxemia and Lieut.-Col. Robert McCarrison, I. M. S., considers the relationship of diet to the physical efficiency of the Indian Army.

The subject of diet is not only all important but fascinating and those responsible for the direction of the *Practitioner* have done a true service in gathering together so many authoritative papers on dietetics. That the vitamines in proper proportions and in adequate amounts are essential constituents of what may be termed "an ideal diet" has been conclusively proven. It has been demonstrated also that certain mineral elements of the diet are just as necessary to good health as iodine, calcium, phosphorus and so on. However, in considering the question of diet the point should not be lost sight of, that the personal equation, climate and occupation all play prominent roles in the choice of food. What is one's man meat is another man's poison and what is good for the tropics is not adapted, or so well adapted, for the arctic region or even for the temperate zone. And occupation, too, is of the first importance. The placing of dietetics upon a more scientific basis is a long step in the right direction, that of prevention of disease. In the near future much more of the chemistry of diet will be learned and what has been long known empirically will be known exactly.

CHRONIC COPPER POISONING

THE large amount of copper on hand and its low price have stimulated the trade to find new uses for the metal. And now comes a report of the experimental work by Mallory[1] on hemochromatosis, which definitely connects that rare disease with chronic copper poisoning.

This has been called the age of copper. It is sprayed on our vegetables and shade-trees, used to kill algae in our drinking water, a necessity in electrical work and common in plumbing fixtures in the form of brass. Food is canned in copper kettles and kept warm in copper steam tables.

Analyses of human tissues have shown copper so uniformly present that the metal has been regarded as a normal constituent of the body rather than as an almost universal contaminant. Careful analyses of drinking water show copper to be present in minute amounts, and the slow corrosion of brass pipes, so common in modern homes, is an added source of the metal.

However, the danger from water and water pipes is slight. Copper is readily attacked by the various acids present in foods, such as acetic, citric, malic, tartaric, and oleic. Consequently any food or drink containing acid will dissolve a certain amount of copper if it comes in contact with that metal.

Hess[2] has shown that milk takes up a slight amount of copper when passed over coils of that metal in the pasteurizing process, and that this may be the reason for lack of vitamine C in pasteurized milk.

The volatile acids readily attack copper, and as many of the illicit stills have copper condensers, moonshine liquor not infrequently contains appreciable quantities of copper. One sample of hooch submitted to Mallory contained 185 milligrams to the liter.

The presence of copper in so many substances taken into the body is rather difficult to reconcile with the rarity of hemochromatosis until we remember that this is a very chronic disease and that the clinical symptoms do not appear until near the end.

Mallory found hemofuesin, the characteristic pigment of hemochromatosis, in 11.6% of 463 autopsies at the Boston City Hospital. In other words, these individuals had ingested

enough copper to produce various degrees of pigment deposit, though not enough pigment had been formed to embarrass the organs and cause symptoms to appear. The production of enough pathological change to give rise to clinical symptoms probably takes years.

There is no doubt that the human organism can take care of minute amounts of copper. Where the dividing line between the harmless and the harmful amount comes we cannot say as yet, and individual susceptibility probably enters in to complicate the problem.

From what is known of the solubility of copper and the amount of it present in various substances, it is fairly safe to say that the danger from copper and brass pipes is negligible, that copper cooking utensils might possibly be dangerous, especially when used for fruits, but that the real risk comes from industrial hazards and from moonshine liquor.

REFERENCES

1 Mallory, F. B.: Am. Jour. Path., i, 1925, 117.
2 Hess, A.: J. A. M. A., 1924, lxxxii, 952-956.

DRUG ADDICTS

Abraham Kossef, M. D., Physician, Clinton Prison, N. Y., has made a study of drug addicts. His report may be found in *The Nation's Health* for January 15, 1925. He finds that of 1460 inmates admitted to Sing Sing from court 5.5 per cent. were drug addicts.

During the last fiscal year the average population of the four state prisons in New York was 4490, of which 5.38 per cent. were drug addicts.

His conclusions are as follows:

1. Only a small percentage of our criminals are drug addicts.
2. A smaller number are imprisoned for crimes involving the use of violence.
3. The number of drug addicts in this country is much smaller at present than has been stated by various agencies, and the number is steadily decreasing.
4. The increase in recent years in the number of drug addicts received in prison is not due to the increase in the number of criminal addicts, but to economic conditions.
5. Drug addiction is not the result of carelessness on the part of the medical profession.
6. Our drug addicts are mostly in middle adult life and have used narcotics for four years or more.
7. The cause of drug addiction is nearly always "association."
8. It is a medical problem demanding police supervision.
9. Treatment should be under custodial care, segregating the criminal from the non-criminal addict.
10. Eradication of the evil will be through proper restrictive laws, and finally substitution of new preparations which are not habit forming, and the absolute discontinuance of the manufacture of our present narcotics.

With an analysis of this large and representative criminal class showing a smaller per cent. of addicts among criminals than has been suspected, the attitude toward this problem may have to be revised.

Dr. Kossef's experience confirms the belief held by some that immediate withdrawal of the drug with appropriate treatment is the logical course to pursue.

MISCELLANY

A Correction

In a paper published by the authors in the March 5th issue of the "Journal" entitled "A Study of the value of Osteopathic Adjustment of the Fourth and Fifth Thoracic Vertebrae in a series of Twenty Cases of Asthmatic Bronchitis" reference is made to a "previous paper" and refers to this paper. The order of publication was reversed through an error on the part of the Journal.

United States Public Health Service

363,063 Cases of Venereal Disease Reported in 1924

An increase in the number of cases of venereal disease reported in the United States in the year which ended June 30, 1924, over the number reported in the previous corresponding year is disclosed by the figures recently made public in the annual report of the Division of Venereal Diseases of the United States Public Health Service. The report indicates that the increase in the fiscal year 1924 amounts to 27,382 cases, or 7.2 per cent. A total of 363,063 cases of venereal disease were reported to the various state boards of health from all sources. This total was composed of 193,844 cases of syphilis, 160,790 cases of gonorrhea, and 8,429 cases of chancroid.

"The fact that the 1924 statistics show an increase over those for 1923 does not necessarily mean that venereal disease was any more prevalent in the United States last year than in the year before," explains the Chief of the Division of Venereal Diseases.

"The greater number of cases now on record at the State boards of health," he continues, "may well be accounted for by the increased efficiency in detecting these maladies and by more conscientious reporting of cases on the part of private physicians.'

During the fiscal year just passed, 504 public clinics reported to the State boards. These clinics treated 118,023 new cases of venereal disease made up of 65,046 cases of syphilis, 49,029 cases of gonorrhea, and 3,949 cases of

chancroid. A total of 2,147,087 treatments were given. The fact that these clinics made 302,152 Wassermann tests for detecting syphilis and 203,008 examinations to discover gonorrhea would seem to indicate that people are beginning to realize the terrible consequences that follow in the wake of these diseases and are willing to take advantage of reputable opportunities for cure.

Reports from 37 correctional and penal institutions were received by the Division. The efforts of those in charge of these institutions have resulted in a large increase in the number of venereally diseased persons discovered and treated. New patients to the number of 7,045 were admitted to treatment in 1924, an increase of 44 per cent. over the year 1923.

The menace of venereal disease is one that is being fought by the United States Public Health Service and the various State boards of health acting in coöperation with municipal health officers. These governmental agencies are trying to impress upon parents, teachers, young people and others the need of wholesome sex education, of prompt medical attention and the necessity for the passage of modern health ordinances and legislation. Among the social institutions which can aid in the fulfillment of this program are the home, the school, the church and the press.

Patriarchs

THE sudden discovery that one may in the not distant future be, as Oliver Wendell Holmes has it, "The last leaf upon the tree in the spring" leads one to look for other leaves in similar case. It will interest nobody to be told that of the 4028 active members of the Massachusetts Medical Society but 27 have been connected with it longer than the writer, but it will surprise many to learn that but 20 of the 4000 were on the rolls fifty years ago.

It may be indiscreet, but I would like to record the names of these twenty men as an encouragement to the youngsters to go and do likewise, for the list consists so largely of those who during a long life have been what might be called superactive, men who have never spared themselves, that one is irresistibly led to the conclusion that hard work is not necessarily fatal and that while it is undoubtedly better to wear out than to rust out the wearing often takes longer than the rusting.

That but two of the long living twenty reside outside of greater Boston and that the oldest retired member, Dr. Lincoln R. Stone, who joined us in 1854, lives in Newton, may be due to that ineradicable dislike to change of residence inherent in the soul of the true Bostonian. It, at any rate, militates against the insulting suggestion that Bostonians live no longer than other people; it only seems so.

Henry P. Walcott of Cambridge stands next upon the list. He joined the Society in 1863. John Collins Warren joined in 1866; William L. Richardson, in 1867; George W. Gay and Edmund H. Stevens of Cambridge, in 1868; Leonard Wheeler of Worcester, Abner Post of Cambridge and William H. Emery of Roxbury, in 1870; Charles A. Lovejoy of Lynn and Elbridge G. Cutler of Boston, in 1872; Fred C. Shattuck, Edward H. Bradford and Russell D. Elliot of Boston, in 1873, as did also George K. Sabine of Brookline and Orlando J. Brown of North Adams; while in 1874 William Appleton, George M. Garland, William S. Bigelow of Boston and George G. Bulfinch and Henry R. Stedman of Boston became members.

S. B WOODWARD, M. D.,
Worcester.

The Complimentary Dinner to Dr. Alfred Worcester

ON December first, 1924, a large number of friends of Dr. Alfred Worcester met at the Hotel Somerset to pay tribute to an honored member of the profession.

The dinner was an interesting and inspiring exhibition of love and respect for one who has taken an honorable and very important part in the progress of medicine in this country. Although circumstances have made it necessary to postpone a report of this meeting, the recital of the substance of the remarks and the full address of Dr. Worcester, even if belated, have not lost much of the moral force contained in the addresses.

The JOURNAL is pleased to be able to put on record this account of a memorable occasion:

Dr. Roger I. Lee acted as toast-master and opened the speaking with the following remarks:

Dr. Worcester and Dr. Worcester's friends: Somewhat over a year ago two excellent young men evolved an idea. That idea was perhaps not original. These two doctors were engaged in a favorite pastime, a very wholesome pastime of theirs, namely, that of admiration of Dr. Worcester, when it occurred to one or the other that Dr. Worcester, having come of age probably before these young men were born, must by the rule of inexorable logic be approaching his seventieth birthday. Now, as a matter of cold, statistical fact, Dr. Worcester's seventieth birthday doesn't come until next June about Commencement time, but that really had nothing to do with their idea. A part of that idea is this dinner tonight. We all know that Commencement time dinners are largely devoted to the birthday celebrations of college classes and students, and hardly is a satisfactory season for birthdays in general. Then, as a matter of fact, the birthday idea is frankly a pretext as far as Dr. Worcester is concerned. It is not because he is about to be 70, but it is because we respect and love him that we are here tonight.

By Dr. Worcester's own expressed wish this

is an assembly of his own medical friends. It would have easily been possible to have filled the largest hall in Boston with Dr. Worcester's friends if we had not restricted the assembly to doctors who are close friends and admirers.

I thought we were to be fortunate in having here as one of Dr. Worcester's friends, Dr. Eliot. Dr. Eliot qualifies to come here because he is the holder of an M. D. degree, because he is a member of the Massachusetts Medical Society, but primarily because he is a friend of Dr. Worcester. But when one is 90, one feels permitted to stay at home by one's fireside in cold wintry weather; and Dr. Eliot sent a belated message just before dinner, expressing his deep regret that he could not be here in person, because he was particularly anxious to do what honor he could to his well beloved friend, Dr. Worcester.

We have included among the profession, unfortunately only for tonight, some of Dr. Worcester's closest friends and closest co-workers in medical causes; and, of course, chiefest among those co-workers is Mrs. Worcester.

A man like Dr. Worcester has friends and admirers everywhere. His influence is not limited to a single locality. It is with particular pleasure that we welcome here tonight from Chicago which always seems a long way from Greater Boston—although they tell me that Boston is quite near to Chicago—that we welcome from Chicago Dr. Dudley and Dr. Woodyatt, and from Baltimore Dr. Kelly.

The toast-master at a dinner like this needs to be nimble and quick in speech, it matters not about his wit. On this occasion the toast-master, so-called, has very little to do. The speakers are all well known and merely have to be announced. The subject of the speaking is also well known, namely, Dr. Worcester. But what a wide range of topics Dr. Worcester affords to the eloquence of these speakers! I cannot help but think how appropriate it would have been had Dr. Cutler of Waltham been spared so that he could initiate the demonstration of regard and of affection for Dr. Worcester. Or perhaps even more fitting, had one been in this place, who has been Dr. Worcester's intimate associate for over forty years. But such is the modesty of men like Dr. Wood that it required persuasion and, I am sure, coercion and possibly some deceit, to get Dr. Wood up here at the head table.

Dr. Cutler and Dr. Wood from their own personal experiences could have recited to you many instances of the many-sided personality of Dr. Worcester. They might have said that years ago Dr. Worcester was a botanist. He taught botany, and due to his persuasive inspiration botanical clubs were formed throughout Middlesex County. I will venture to say that there are other illustrations of the many-sided personality of this man; and a great many of those illustrations are quite unrelated to medicine. But in this gathering of doctors we acclaim loudest of all what Dr. Alfred Worcester has stood for and has accomplished in the merciful calling of medicine. Paraphrasing an ancient saying which seems to me to be peculiarly appropriate, "the patient finds in Dr. Alfred Worcester, as he has always found, a cure perhaps, relief probably, but comfort always."

It is fitting that the first speaker should be a Waltham boy well known to Dr. Worcester in his early days, the early days of the boy in Waltham. He is a graduate of one of our great medical schools in this vicinity and he has been for some years a professor in another great medical school in this vicinity—Dr. Timothy Leary, formerly of Waltham, now of Boston.

Dr. Leary began his speech by referring to Waltham, the site of the activities of Doctors Fernald and Worcester, as his birthplace and emphasized two outstanding characteristics of Dr. Worcester, dealing first with the pioneer spirit of the honored guest who is a descendant of a race of pioneer preachers. The first evidence of this pioneer quality was shown early in life by his introduction to the Lying-In Hospital, where he was an intern, mercuric bichloride, to the consternation of his Chief, and the institution of the practice of written orders and written records. His next demonstration of the pioneer spirit was in the creation of the Waltham Hospital and the Waltham Training School for Nurses where his conceptions of a community hospital and the best possible preparation of nurses for service in the homes of patients could be applied. The results have demonstrated originality in and understanding of the application of the art of medicine and nursing to the needs of the average sick person, and have met wide approval.

Dr. Leary paid merited tribute to the influence of Dr. Worcester in advancing a correct understanding of the treatment of appendicitis and his influence in promoting the plan for the creation of state sanatoria for the treatment of incipient tuberculosis; even though Dr. Worcester later modestly proclaimed denial of the credit for this and also the honor of establishing the Victorian Order of Nurses. It is, however, common knowledge that if it were not for his efforts with Canadian physicians and the work of Miss McLeod under his inspiration, the efforts of the Countess would have been in vain. These are some of the important activities of this pioneer. Minor matters of medical interest in which he has led the way would fill a volume.

Under the theme of "Worcester, The Crusader," Dr. Leary referred to the courage of Dr. Worcester in attacking abuses, citing his efforts to reëstablish confidence in the natural process of parturition in contradistinction to the belief on the part of some women and doctors that pregnancy and delivery are forms of disease which have led to meddlesome midwifery "which in some cases lacked only a cash register to make it a well organized business" when surgery was

offered as a standard choice to delivery per vias naturales. The higher mortality following this orgy of operating prompted investigations within the profession under the leadership of Dr. Worcester with the result that adherence to more normal management of patients brought about lower mortality rates. The influence of James Mackenzie in rejecting the more mechanical methods of study and treatment was used in illustrating the same traits in Dr. Worcester tending to reëstablish the standing of the general practitioner and diminish the encroachments of specialism to the end that there will be more general adoption of those qualities which have caused the world to rise up and call our profession blessed.

In introducing Dr. Howard Kelly of Baltimore, Dr. Lee said: There is always a strong attraction between kindred spirits. The next speaker is one of the kindred spirits with Dr. Alfred Worcester. He too is versatile. He too is charming. He too is full of fire and enthusiasm for many a worthy cause irrespective of its popularity. He too has been touched by that spark which elevates the few above the many, and withall he has been preëminent in his chosen specialty, and for twenty years he was a professor in the Johns Hopkins Medical School. Perhaps this kindred spirit can give a sympathetic appreciation of his friend, Dr. Alfred Worcester—Dr. Howard Kelly of Baltimore.

Dr. Kelly expressed his pleasure in being given the opportunity to voice his sentiments and, in looking about the audience and seeing Dr. E. C. Dudley, felt that all states and lands would have sent their tributes had an opportunity been afforded. Speaking of having escaped an analogous experience he felt sorry for Dr. Worcester's being subjected to this trying ordeal but which not being perfunctory has given to Dr. Worcester's friends the privilege of expressing the outpouring affection and esteem for one who has been an inspiration. The especial thought in Dr. Kelly's tribute was the good that may be carried to one in distress must be an expression of heartfelt sympathy which is an index of the spiritual side of one's nature. "The world is starving today not for big heads but for sound heads controlled by warm hearts." This Dr. Kelly felt is so important in our relation with others that directions have been given to his secretary to slip a little piece of heart into his correspondence. Dr. Worcester too has practiced the art of giving full play to the expression of heartfelt sympathy.

Referring to the great advances in medicine, Dr. Kelly congratulated Dr. Worcester in having come into the world at a fortunate time when he could be in the front ranks of those who have contributed so much for humanity. Speaking of the scientific treatment of appendicitis, Dr. Kelly said, I like to think in pictures, and there rises before me a splendid monument on Commonwealth Avenue, with Fitz standing on the highest step, thesis in hand, declaring to the world that the case is clear, the diagnosis can be made, and the surgeon must act; while stepping up with eager uplifted countenance comes Alfred Worcester, triumphantly exclaiming, "What you say, Fitz, is true, for I have already done it," dramatically presenting the big sausage-shaped recently removed appendix. Another inspiration received from Worcester came during a visit to the Waltham Hospital when Dr. Worcester in his work with nurses demonstrated Florence Nightingale's stand for the highest professional ideals in the nursing profession with Christian faith, ethics, and heart above all else. Dr. Kelly affirmed, I wish we had more Worcesterian training schools today; they must come or the situation is lost.

Dr. Kelly then paid his respects to Mrs. Worcester in these words: Lastly, nay, rather first of all, the greatest asset in Alfred Worcester's life is the one which is least conspicuous,—a noble lady in the home, a veritable bishop coadjutor, who through these long and sometimes difficult weary years has stood by him in his splendid work, coöperating to the limit and beyond, stimulating, advising, and even bearing with some of those vagaries always inherent in genius—a true helpmeet, the really God-given better part of every man, though we often mention it lightly. God bless her for all we owe to her inspiring presence. Were it not for her, I rather opine we should not be here tonight. Let me then in closing demolish my Worcester-Fitz monument to erect yet a nobler one, that of an ideal husband and wife grouped lovingly, looking bravely out over the sea of suffering humanity and there discerning their common task as they proceed eagerly to embrace all the opportunities offered by their calling in rendering jointly their glad life's service—America's best and highest ideal, the realization of the primal blessing "and they twain shall be one flesh." Had I papal power, I would canonize them among Boston's patron saints, and were I gifted with supernal power I would (indicating two circles with the finger) inscribe an aureole over the head of each.

Dr. Lee then introduced the next speaker as follows: Dr. Richard Cabot has not always been gentle or always complimentary to the doctors, but no one, as far as I know, has ever questioned Dr. Cabot's sincerity or courage. Perhaps he has seen the practice of medicine from a different angle than most of us. Recently, and unfortunately I believe for the medical profession, his other duties have overshadowed his medical activities. However Richard Cabot, Professor of Social Ethics, idealist, apostle of the truth, always will be an outstanding, distinguished physician—Dr. Cabot.

Dr. Cabot paid especial attention to the care

of patients exhibited by Dr. Worcester which was so well known that it has been reported that a movement was under way at one time to ask Dr. Worcester to accept a professorship under the title of "Professor of the Care of Patients."

Dr. Cabot expressed regret because of the too rare occasions when he could meet Dr. Worcester in the sick room because Dr. Worcester "was not over fond of consultants like myself or specialists like you." He knew too well that the general practitioner has to stay behind and look after what they left for it is necessary to controvert the harm when a patient is told that a consultant is needed and further, the effect of a long discussion and explanation of the diagnosis and pathology in a given case, so that the attendant may have to begin all over after the consultant has gone and reëstablish hope and confidence. An especial feature of Dr. Worcester's practice was freedom from therapeutic nihilism and the free use of appropriate remedies. Another important quality of his practice was the maintenance of an atmosphere favorable to recovery consisting especially of hope, faith in God and an affectionate charity between all those around the patient. Speaking of the length of Dr. Worcester's visits to his patients, Dr. Cabot explained that, although a busy practioner, an abundance of time was given to every case in order that all direct and coördinate features of the illness should receive adequate consideration. These visits might last hours or weeks, one lasted nearly a year. Especial mention was made of the painstaking care of patients who are about to die. Dr. Cabot closed his remarks by referring to the general practitioner in these words: "We have seen a great deal in these latter years pointing rather in the opposite direction,—suggesting that the general practitioner is going out. I have asked myself: Why is the general practitioner going out when people want him more than ever? and I have thought that the answer was: Because there are not enough men alive of Dr. Alfred Worcester's stamp. But if the general practitioner is coming back, it is because the medical profession is beginning to recognize true greatness and to line up behind Dr. Worcester as we all of us do tonight."

The next speaker was Dr. Vincent Y. Bowditch, who responded to the toast: How happy is Dr. Worcester in his friends! This occasion would certainly not be complete without a word from one of Dr. Worcester's old friends, Dr. Vincent Y. Bowditch. These two men, both idealists, both enthusiasts, had visions and dreams, and working together shoulder to shoulder they saw those visions and dreams established in the history of tuberculosis in Massachusetts and in the United States—Dr. Bowditch.

Dr. Bowditch opened his remarks by the quotation, "From the fullness of the heart the mouth speaketh," and said, It would be impossible for my mouth to utter all that is in my heart on an occasion like this when we are doing honor to this man. No one in this assembly, perhaps he least of all, realizes the influence he has had upon my life in the last thirty-five years.

He then outlined a brief history of his association with Dr. Worcester in the building of the Rutland Sanatorium under State authority after having learned through his work at Sharon that consumption could be successfully treated in this harsh, inclement climate. Dr. Worcester was largely instrumental in changing the plans to make Rutland an asylum for hopeless cases to the more useful effort of treating tuberculous cases in the incipient stages when there was a possibility of recovery. This first state sanatorium for this type of the disease in the Union was due in large measure to Dr. Worcester.

Dr. Bowditch closed with this tribute: Such is the man whom we honor this evening. Others have spoken of his humanitarianism. Nobody knows it better than I but I must not keep you longer.—Alfred Worcester, brother in the profession, brother in spirit, I salute you. May you be spared to us many years to be the inspiration and comfort to the many who love and honor you.

Dr. Lee then introduced Dr. Dwight O'Hara who spoke as follows: Dr. Lee, Dr. Worcester, Ladies and Gentlemen: What Dr. Lee has just said is perhaps the truth, but it is not the whole truth because at every turn we have sought Dr. Lee's advice in making our plans—and I will say that his advice is worth seeking. Those of us who arranged this dinner have been having a wonderfully good time. The pleasure of our task has been partly due to our trusting belief in Dr. Worcester and in all that he has stood and is standing for, and partly to the spirit and enthusiasm of the gentlemen in this room. But the demonstration does not stop at the doors of this room. I have here a package of letters from some of those who are unable to be with us tonight, and as an expression of their loyalty I am now going to present these letters to Dr. Worcester.

And, Sir, I have here another expression, though a very feeble one, of the love and affection of every man in this room for you. (At this point copies of the book, *Alfred Worcester, 1925*, containing some of the essays written by Dr. Worcester, were distributed to those who were present.)

The closing address by Dr. Worcester was prefaced by Dr. Lee in these words: We are going to give Doctor Worcester just a minute to refresh his memory. In this book are, of course, some of the writings of Dr. Alfred Worcester. I am sure he will agree with us as he reads his own writings that they are just as good, and that they are just as fresh now as they were the day they were written, and some of

them, mind you, were written 40 years ago.

Doctor Worcester, what has been said here tonight has been far less than the whole truth, far less of honor, far less of affection. Would that this assembly could designate you by some fitting title that would raise you above the generality of men and of physicians, that could make it known to all people that you have lived a fuller life, a more helpful life than the rest of us. Yet you have by your own life given to the title of "doctor" a deeper and a more noble meaning. As has been so often said here tonight, you are yourself an inspiration for the medical profession, and the medical profession is better for what you have stood for and for what you have accomplished. As we have been sitting here tonight, Dr. Leary has passed me a card upon which this is written, "Dr. Fernald who expected to come to this dinner and who expected to enjoy this dinner just as we all have enjoyed it in honor of Dr. Worcester, one week before his death expressed publicly at a dinner, taking as his theme the coming Worcester dinner, two thoughts—first, If we had had more doctors like Worcester, Christian Science never could have gotten started, and other cults would have died unborn; and secondly, the amazing influence of Doctor Worcester for good among young men has been wide-spread." Dr. Fernald said he knew of no man who was so powerful an influence for good in the whole medical profession.

But let us be done with words, let us even put off until tomorrow the reading of this notable little book of Dr. Worcester's; let us rather than the spoken or printed word have the real man, the real physician himself, Doctor Alfred Worcester.

In responding, Dr. Worcester said. Dear Doctor Roger Lee and dear friends: It is not safe to believe more than half of what one hears. For example, the heart specialists say that there is no such thing as acute dilatation, but if their fluroscopes were trained on me at this moment, they would find mine half way up to my mouth; and if I believed more than half of what I have heard from this table, they would find it still higher, and I should not be able to utter a word. But I would not have you believe that I doubt the veracity of those who have spoken. To do that in one instance would be to accuse a dear friend of not practising what he preaches. I do not doubt they believe what they have said, or most of it, but they have made this great mistake, they have mistaken me for what I have wanted to be; and so in the portrait they have painted for you there my best foot is too far forward, and they have too kindly hidden my short-comings and deficiencies. But I do not want to spoil their picture and so I shall continue their pleasant treatment of the subject; for it will be more enjoyable for you as well as for me if I speak of my ideals rather than of my achievements.

Deeply grateful as I am for the honor paid me, I am most grateful for it as a recognition of the work of a general practitioner, for to tell the truth we of that denomination are in sad need of encouragement. Our ranks are fast thinning, recruits are few and far between. In fact the country doctor is already a rare bird, hardly ever to be found nowadays except in remote rural regions beyond the reach of the specialists' limousines. The reasons for his disappearance are plain enough. Had the privileges of the general practitioner been brought to the attention of medical students, had the art of medical practice as well as the science been taught them, no doubt there would have been some in every medical class who would have striven to acquire the art and then would have chosen general practice as the largest field for it.

Not from any premeditated design was the family doctor and the art of practice consigned to oblivion. It was only the natural result of modern medical education. What other product than specialists can be expected of a medical school where all the clinical instruction is given by specialists? Again with the tremendous advance of medical science in the last half century it is impossible for any man to become proficient in all of its branches, and moreover even a tolerate competence for general practice is obtained with more difficulty than proficiency for a specialty. This of course is a deterrent to an ambitious student. Then again there is the economic reason. General practitioners, somewhat overawed by the superiority of the specialists, never charge for their services, even when sure of their competence, more than a half and often not a tenth of the specialist's fees. This is inevitable because the general practitioner's employment is more and more with families who cannot afford the specialist's fees and this again acts as a powerful deterrent to ambitious young men. But those of us who have clung to general practice have been rewarded far more richly than with gold. We have found in it the largest possible opportunity for fellow service, and there is no greater privilege than that; and again in no other calling can one so easily win the love and confidence of one's neighbors.

No physician who is interested in only one organ of the body and much less one who is intent upon only one of its diseases can really know his patients or be known by them. Here is where the general practitioner has an enormous advantage. He has to think of the whole body with all its power, with all its frailties, and more than that, he of all men, seeing life come in and go out of this world, is best enabled to realize the essential nature of our existence, which is not that we have souls, but that we are souls and have our earthly bodies for a brief moment of time.

Again the general practitioner, the family physician has the opportunity of knowing his

patients not only as individuals but as members of their families. In many cases successful treatment depends on the coöperation of the family to prevent the depressing influence of their anxiety, and it is of still more avail to enlist their encouraging influence. It is no small advantage in the care of a patient to have known his parents and perhaps his grandparents.

It was my good fortune to succeed to the practice of two great general practitioners. The elder Horatio Adams took care of me when I was born and until I could appreciate his kindness. His widow and his son, Dr. B. F. D. Adams, were my counsellors when I began to practice; and the value of this counsel was inestimable. I have taken care of some families for four and in one instance for five successive generations; and it so happened that in her later years I took care of the first patient that Dr. Horatio Adams had one hundred years ago. Waltham air is salubrious. I have known six women in my neighborhood who passed their hundredth year. I wish I could say they were all of them my patients; but one was addicted to homeopathy, and one of them never indulged in any kind of medical treatment.

In the references that have been made to my life work—and I think it has been pretty well covered this evening—as mention has been made of my habit of staying with the dying I feel like giving my reasons for so doing. It was the old fashioned way. Doctors were supposed, as Dr. Oliver Wendell Holmes said, to take care of the coming in and going out of life; but lately only the first part of that duty has seemed to remain upon the doctors' shoulders and we know how obstetrical science has advanced. It is quite true that when I was in the Lying-In Hospital forty odd years ago the patients were dying right and left of septicemia. I remember at one time there was not a single patient in the hospital that was not suffering from puerperal fever; but the credit of bringing corrosive sublimate into that hospital belongs to Rufus Kingman who had just returned from Prague and told us that septicemia had been eliminated there by the doctors scrubbing their hands in solutions of corrosive sublimate. By that procedure septicemia was stopped at the Lying-In Hospital.

There have been other great advances in obstetrics, but what has been done to increase our power to give comfort to the dying? Almost nothing. And yet there is much that can be done for them long after they are able to make known their wants. I wonder if young doctors read Dr. Edward Hammond Clark's "Visions." It is a wonderful book which ought to be a required text-book in medical schools. I want to have a doctor stand by me when I go, and therefore I consider it my privilege to stand by patients who have entrusted me with the care of their lives. The fact is that this duty like many others has been transferred to the nursing profession, and I want to speak of this transfer. If we could be sure that all of our patients have as good care as some of them have we should be more justified than we are in this abdication. We know that such is not the case and we know that good nursing service is not obtainable by the majority of the families living about us. It cannot be afforded. And a doctor has not done his duty when he gives the telephone number of the nearest nurses' registry for a nurse to serve as his executive during his absence. The *cure* of the sick is our business, is it not? And the cure of the sick depends more largely on the *care* of them than on anything else. Therefore the care of the sick is our responsibility which can rightly be transferred to a nurse only when we know the nurse will carry out our directions and do as well for the patient as we can do ourselves. That was my conviction when I persuaded my colleagues to join me in starting the Training School in Waltham.

I still believe that as nine tenths of nursing service is given in private families it is for that service that nurses should be especially trained, and I know they can be trained *for* that service only *in* that service where of course they must have proper supervision. But in carrying out this principle our Waltham Training School has met with opposition from the leaders of the nurses' organizations. Those ladies believe that a hospital training fits a nurse for any kind of nursing. Formerly they did not think the Waltham nurses were fit associates because part of their training was preparatory. Then they adopted that. Next they objected to the Waltham nurses because part of their training was given them in district visiting service and afterwards they adopted that; and yet we cannot satisfy them. Always some new requirement is thrust upon us. The last criticism is that our pupil nurss do not wear the same colored stockings. We are told that unless our nurses wear white stockings in summer and black stockings in winter, they will not be considered fit for registration in Massachusetts. I would not waste your time with such petty matters, were it not for the opportunity it gives me to testify to the hearty support given us from the medical profession since our school began, and also to show why we need a continuance of that support.

As I look back over my life work, I realize keenly how great my debt is to others. It seems as if I had done nothing but act as though what I have received from others were my own. I think first in this connection of my mother who was a minister's wife and a famous neighborhood nurse. That was before nursing service could be hired in the market even by the rich. It was she who taught me the joy of helping the helpless. Before I was sixteen years old she sent me out as a night watcher to a school boy sick with typhoid fever. I have had many a less valuable lesson since that night. When I began

my practice I occasionally had her help with the new-born and with the dying, and the memory of her tender ministrations is a never failing inspiration. And may I not in the intimacy of your friendship make acknowledgment also to one in whose close companionship I have found my ideals and aspirations ever rising to still loftier heights, and yet so helpful has been her clearer intuition at critical turnings of the trail, so dear her encouragement at the steepest and darkest places, that the upward climb though lengthened has seemed shorter,—shorter even than it seemed to the unpracticed eye of youth—so mighty is the working of perfect love. But I fear she will hardly pardon even this inadequate tribute, and so may I not with your approval send to her these roses which belong so much more to her than to me.

In dwelling upon the longer preparation needed for general practice I am prompted by my own experience. My own medical education was scandalously scanty. I was in the Harvard Medical School for only ten months, and this is how I did it:—It was possible forty odd years ago to go up for examinations without having attended the lectures, and I audaciously thought I could employ my time to better advantage otherwise. So instead of going to lectures I looked over my fellow students' note-books a few days before the examinations. The first year I escaped by the fiction of registering with a doctor as his student ,and spent it in work at Cambridge for which they gave me my master's degree. The second half of the second year I escaped by a fortunate dissecting room erysipelas for when on my back as a sick man I learned more about the practice of medicine, what should be done and what should not be done, than I could have learned in the lecture courses. And the last half of my third year I escaped by my interneship in the Lying-In Hospital. It happened to be closed at the time of the final examinations, and I went up thinking it would be good practice. I was never more surprised in my life than to find my name printed as a candidate for the M. D. degree. I went out to Cambridge with fear and trembling, and not until President Eliot handed me my diploma was I sure that I would not be tried for high treason.

This lack of education would have been a greater handicap if I had started independently. But my first years of service were as a junior under one of the finest men I have ever known, Dr. Edward R. Cutler. It was he who backed me up in my first appendectomy to which Dr. Kelly has referred.

I never knew until months afterwards what I had fished out of that little girl's belly in the summer of 1886. I never reported the case, not from modesty but from mortification, but this evening I am going to. The little girl was sent out from the Massachusetts General Hospital to die. The diagnosis of the surgeons there was a psoas abscess filling the right half of the belly, and operation was deemed impossible because of the child's septic fever. I had already seen several deaths from what used to be called stoppage and was beginning to be called peritonitis. I had made several autopsies on cases that had died after the customary medical treatment of the day, which was "mixed treatment" for some gave opiates, some gave salts, and some gave both, alternately or in combination. I made up my mind that never again would I let a patient die of such a disease without giving the foul pus at least an antemortem exit. My test came with this litlte girl, for whom there was "no hope" as the Massachusetts General surgeons said. Then I was up against my determination and so I said I would not have the care of her if I could not operate. The family reluctantly consented, and Dr. Cutler backed me up. This is what I did: I plunged a trocar through the loin which brought foul pus. With this as a director I cut my way for two fingers into the abscess cavity and found what seemed like a sausage hitched by one end, grabbing it with a double tenaculum I pulled it out. The patient is still living.

Shortly after that I helped Dr. Cutler do his first appendectomy. Not yet daring to take the straight road, we burrowed in under Pourpart's ligament and pulled out another gruesome object which certainly was not vermiform; and Dr. Fitz had not published his treatise calling world attention to the fact that the appendix is the root of all evil in that locality.

In the next case I was sure what was the matter from the first, but it was not until after a week of misery that I woke up from my morphine sleep to see Dr. Maurice Richardson and Dr. Elliot and somewhat to my dismay, Dr. Fitz, whom I had never seen before except at autopsies. I prayed for the operation and I owe my life and so you owe this meeting to Dr. Elliot's intrepidity in operating against the advice of the others who believed I was too far gone. While I was on my back I urged a neighbor of mine to submit to early operation so that he might escape the misery I had been suffering; and when Dr. Cutler and Dr. Wood brought back that angry red appendix, and the man was able to recover, before I was off my back, I made up my mind to urge the immediate operative treatment; but I had no idea of the opposition I was to encounter in the next few years. That, however, belongs to the forgetable past.

I know I am keeping you too long, abusing your patience, but I must clear myself of taking credit which in so large part belongs to others. For instance, I do not refuse some share of the credit for having established the first State sanatorium for the cure of the tuberculous instead of having it, as was planned by the Legislature, a hospital where consumptives were to go to die. But in so doing I was merely fol-

lowing in the wake of the very man who tonight praises me for doing it. Why! the success at Rutland, as the success at Sharon, was due to the self-sacrificing devotion and enthusiasm of Vincent Y. Bowditch.

And in your kind appreciation, dear Doctor Leary, you have exemplified the great advantage the family physician has in knowing the children from the very beginning and in being loved by them. Sometimes the children's confidence is embarrassing. I remember once a contest I had with President Eliot as to which of us had ever received the greater compliment. After he told me the greatest he ever had, I told him of the telephone summons I once had from a dear little boy who begged me to come quick because his rabbit was dead.

As for Richard Cabot, why, I have learned more from his mistakes in diagnosis, which he has published with such admirable candor, than I have from the lavish broadcasting of many another's successes.

And I don't suppose you, dear Howard Kelly, ever dreamed of what it was for an amateur surgeon to see your wonderfully skillful work and read your lucid descriptions. Without the latter, I should not have succeeded with my first Caesarian operation.

As for you, Roger Lee, and many others here tonight, I am taking your wisdom every day and passing it on as if it were my own.

I don't propose to pay all of my debts. Time would not allow that; but I should never forgive myself if after receiving the honor you pay me this evening I did not tell you how for fifty years through every storm Henry Austin Wood has stood at my shoulder. Many a time he has saved my patients from my blundering by his surgical skill, and I know no better surgeon. My only grievance has been a constant pricking of my conscience by his greater diligence, and faithfulness, which I have been able to excuse in him only because of our love for each other that has never suffered the least interruption.

Some of you gentlemen only lately have made the acquaintance of Quimby Gallupe and Dwight O'Hara. To have such support from the generation following is more than any man could reasonably expect. It is more of a blessing than it seems as if we have a right to have as the world goes. Words fail me to describe their loyal support, their readiness to help at every turn; and their confidence—until the last few weeks when, as I came into the room, they would shove things out of my sight so that I felt I was interrupting in some vital matter. I knew Dr. Gallupe was happily married and I had hope for Dr. O'Hara, and so imagined there was something in the wind. But now I believe it was this book they have presented me,—I have no doubt I shall read it (as the engraved cards say) with interest and profit. But I shall never know how to thank them. If my patients knew what I owe to them, I should surely be shelved.

Many years ago after a lovely afternoon with Florence Nightingale as I rose to go she asked me to tarry for a moment while she asked God's blessing upon "our work"; and now, dear friends, you who have so kindly listened, after thanking you all, may I not ask you to pause for one moment to think of the great Physician of our souls—while I pray that His most bounteous blessings may be yours, and that He may help us all in our work.

The Massachusetts Medical Society

MEMBERSHIP CHANGES AND CORRECTIONS OF THE DIRECTORY

JANUARY 1 TO MARCH 1, 1925

Alphabetical list compiled by the Secretary from the official list

Abbe, Alanson J., from St. Cloud, Fla., to Winter Park, Fla. Name placed on retired list by Council, Feb. 4, 1925.

1909 *Adamian, Parnag Adam*, died at Worcester, Feb. 3, 1925, aged 56.

Aronson, Charles, from Salem (Essex South) to Los Angeles, Calif. (Non-resident List), 3556 Eagle St.

Babcock, Harold L., Dedham. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office Boston, as before.

1924 Baehr, Frank Herman, Boston. Boston City Hospital. (Name omitted from Alphabetical List by mistake.)

Bailey, Walter C., Cambridge. Transferred from Middlesex South to Suffolk by Council, Feb. 4, 1925. Office Boston, as before.

Barron, Maurice E., Boston, from 484 to 475 Commonwealth Ave.

Baxter, Clarence Pennell, San Diego, Calif. Resignation accepted by Council, Feb. 4, 1925.

Bigelow, Edward B., Worcester. Change "1" to "9," Walnut St.

Blair, Orland Rossini, Clark's Summit, Pa. Resignation accepted by Council, Feb. 4, 1925.

Bloom, Robert R., from Boston (Suffolk) to Ossining, N. Y. (Non-resident List), Ossining Hospital.

Bogan, Isabel K., from Beverly (Essex South) to Brookline (Norfolk), 193 Aspinwall Ave.

†1877 *Booth, Edward Chauncey*, died at Winter Park, Fla., Jan. 18, 1925, aged 75.

Brennan, John Patrick, Camden, N. J. Resignation accepted by Council, Feb. 4, 1925.

Boutwell, Horace K., Brookline, 15 Green St. Change from Suffolk to Norfolk. (Original transfer, in 1916, was an error.)

†1884 *Broidrick, James Patrick*, died at Jamaica Plain, Feb. 26, 1925, aged 76.

Cahill, Henry P., Jamaica Plain, office now Boston, 514 Commonwealth Ave.

Capeles, T. F., Haverhill, from 191 Merrimack St. to 208 Mill St.

Champion, Merrill E. Change "Brockton" to "Boston" in Alphabetical List. (A typographical error.)

Cochrane, Robert C., Brookline. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.

Cohen, Milton M., Roxbury, from 40 Gaston St. to 8 Waumbeck St.

Coleman, Robert M., from Boston (Suffolk) to Wellesley (Norfolk). Office Boston, 270 Commonwealth Ave.

Cunning, Daniel Sylvester, New York City. Resignation accepted by Council, Feb. 4, 1925.
Coolidge, Sumner, Plymouth. Resignation accepted by Council, Feb. 4, 1925.
Cooney, M. Blanche, Haverhill, from 67 Winter St. to 23 Vine St.
Drake, William Abram, No. Weymouth. Name placed on retired list by Council, Feb. 4, 1925.
Fallon, John M., Worcester, now has office at the Peter Bent Brigham Hospital, Roxbury.
Fipphen, Earl E., Worcester, from City Hospital to 21 West St.
Fisk, Arthur Lyman, New York City. Resignation accepted by Council, Feb. 4, 1925.
Fitzgerald, James B., Boston, from 150 St. Botolph St. to 393 Massachusetts Ave.
Fremont-Smith, Frank, Jr., Cambridge. Transferred from Middlesex South to Suffolk by Council. Office the same.
Gaunt, George A., Worcester. "State Hospital," not "City Hospital."
†1899 *Gavin, John Harrison*, died at Roxbury, Dec. 9, 1924, aged 71.
Ghormley, Ralph K., Brookline. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.
Good, Frederick L., Brookline. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.
1902 *Goodridge, Frederick James*, died at Cambridge, Jan. 15, 1925, aged 51.
Granger, Frank Clark, Randolph. Name placed on retired list by Council, Feb. 4, 1925.
Griffin, Charles H., from Boston (Suffolk) to New Bedford (Bristol South), 101 School St.
Halback, Robert M., from New Bedford (Bristol South) to Madison, Wis. (Non-resident List), Madison General Hospital.
Halloran, M. J., from Jacksonville (Non-resident List) to Worcester (Worcester), 42 Green St.
Hodgdon, Ralph F., Winchester. Transferred from Middlesex East to Middlesex South by Council, Feb. 4, 1925. Office the same.
Holtt, Eugene Gorham. Name placed on retired list by Council, Feb. 4, 1925.
Howard, Charles T., Hingham Center. Transferred from Norfolk South to Suffolk by Council, Feb. 4, 1925. Office the same.
Howard, Hubert H., Brookline. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.
1911 *Hudnut, Paul Albert*, died at New York City, Jan. 24, 1925, aged 56.
Hughes, John, from Holyoke (Hampden) to Pittsfield (Berkshire), 230 North St.
Huntington, James Lincoln, Boston. Change "29" to "24" Marlborough St. (Typographical error.)
Hurley, John Joseph, Boston. Resignation accepted by Council, Feb. 4, 1925.
Irwin, V. J., Sr., †1923, Riverview, Fla. Irwin, V. J., Jr., Springfield, 274 Main St. (Please correct in Directory.)
Jellis, Walter, from Somerville to West Somerville. Same address.
1919 *Jordan, Michael Matthew*, Worcester, died in 1923, aged 78.
1889 *Keegan, Charles Andrew*, died at Arlington, Jan. 15, 1925, aged 59.
Kissock, Robert J., New York City. Resignation accepted by Council, Feb. 4, 1925.
Leavitt, Frank C., Belmont. Resignation accepted by Council, Feb. 4, 1925.
Lee, Harry Jason, Lexington. Transferred from Middlesex South to Suffolk by Council, Feb. 4, 1925. Office the same.
Litchfield, William Harvey, Marblehead. Name placed on the retired list by Council, Feb. 4, 1925.
1895 *Lougee, Frank Taylor*, died at Lynn, Jan. 6, 1925, aged 62.
MacMichael, Earle H., Englewood, Fla. Resignation accepted by Council, Feb. 4, 1925.
Macnaughton, Elizabeth, Brookline. Office now Boston, 483 Beacon St.
1898 *Maguire, Charles Francis*, died at Somerville, Feb. 28, 1925, aged 52.
Marcus, S. M., from Beverly to Peabody, 67 Main St.
Marshall, John R., from Somerville to West Somerville. Address the same.
McCarthy, Louis F., Malden, from 56 Summer St. to 11 Chestnut St.
McEvoy, Thomas E., Worcester, from 37 Portland to 718 Main St.
McLaughlin, Joseph H., from South Boston (Suffolk) to Dorchester (Norfolk), 799 Columbia Rd.
Moir, Marguerite W., from West Roxbury to Boston, 465 Beacon St.
Monette, Camille J., from Taunton (Bristol North) to Willimansett (Hampden).
Moore, Frederick P., from East Norfolk (Norfolk) to Watertown (Middlesex South), 258 Belmont St.
Morse, George W, Boston, from 375 to 475 Commonwealth Ave.
Munro, Donald, Milton. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.
Murphy, William Parry, Jamaica Plain. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.
Naurison, J. Z., Springfield, from 172 to 235 Main St.
1899 *Newton, Aaron Lewis*, died at Brookline, Jan. 24, 1925, aged 59.
Overholser, Winfred, Wellesley Hills. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Office the same.
Packard, Fabyan, from Graniteville to Tewksbury, State Infirmary.
Packard, Horace, Boston. Name placed on the retired list by Council, Feb. 4, 1925.
Palmer, Ezra, from Dorchester (Norfolk) to Boston (Suffolk), 182 Bay State Rd.
Patch, William T., Roxbury. Boston office should be "144" Commonwealth Ave.
Perry, Sherman, from Tewksbury (Middlesex North) to Winchendon (Worcester North), 20 Walnut St.
Petrillo, Carmen R., from Boston (Suffolk) to Everett (Middlesex South), office Boston, 20 Cooper St.
Phipps, Cadis, from Boston (Suffolk) to Brookline (Norfolk), office Boston, 587 Beacon St.
Pfeiffer, Albert, from Boston (Suffolk) to Albany, N. Y. (Non-resident List), Public Health Service, Capitol, Div. of Venereal Dis.
Price, O. J., from Somerville to West Somerville. Address the same.
Randall, Francis D., Tampa, Fla. Name placed on the retired list by Council, Feb. 4, 1925.
Ragle, B. H., from Brookline (Norfolk) to Boston (Suffolk), 226 Marlborough St.
Raskin, Nehama, from Waltham (Middlesex South) to Taunton (Bristol North), Taunton State Hospital.
Ripley, Horace G., Brattleborough, Vt. Resignation accepted by Council, Feb. 4, 1925.
Ripley, William L., Brighton, from "19" to "9" Oak Sq.
Rock, John Charles, Boston. Delete "Charles." Name now is "John Rock."
Rothwell, Charles R., South Boston, from 573 to 517 Broadway.
Russman, Charles, from Lawrence (Essex North) to Middletown, Conn. (Non-resident List), Connecticut State Hospital.
Sartwell, R. H., from Cranston, R. I., to Howard, R. I., Rhode Island Infirmary.
Shafer, Rudolph J., King's Park, L. I. Resignation accepted by Council, Feb. 4, 1925.

Shea, Francis X., from Roxbury (Norfolk) to Everett (Middlesex South), 519 Broadway.

Shoemaker, Amzi B., from North Attleborough (Bristol North) to Boston (Suffolk), 7 Marlborough St.

Shortell, Joseph H., Newton Center. Transferred from Middlesex South to Suffolk by Council, Feb. 4, 1925. Same address of office.

Sibley, Benjamin H., Boston, from 375 to 475 Commonwealth Ave.

Silver, Louis S., Malden, from 230 to 250 Salem St.

Sims, Frederick R., from Melrose (Middlesex East) to Augusta, Ga. (Non-resident List), U. S. Veterans' Bureau Hospital No. 62.

Stein, Louis C., Watertown. Transferred from Middlesex South to Suffolk by Council, Feb. 4, 1925. Office as before.

1897 *Stevens, Henry Burt*, died at West Roxbury, Jan. 31, 1925, aged 55.

Stevenson, Willis M., North Easton. Transferred from Bristol North to Plymouth by Council, Feb. 4, 1925. Address the same.

Stone, Moses J., from Rutland (Worcester) to Dorchester (Norfolk), 109 Ellington St.

Suarez, Jemaro, Porto Rico. Resignation accepted by Council, Feb. 4, 1925.

Tartakoff, Samuel, from Taunton (Bristol North) to New Bedford (Bristol South), 248 Pleasant St.

Taylor, J. Richard, Fairhaven, from 348 to 313 Main St.

Tibbetts, James T., Mineola, N. Y. Name placed on the retired list by Council, Feb. 4, 1925.

1893 *Tirrell, Vinson Meader*, died at Westborough, Jan. 18, 1925, aged 65.

Townsend, Charles W., Boston. Name placed on the retired list by Council, Feb. 4, 1925.

Turnbull, John A., Brookline. Transferred from Norfolk to Suffolk by Council, Feb. 4, 1925. Address the same.

Ward, George O., Worcester, from 875 Main St. to 6 Ripley St.

Wheeler, Lucia, from Wernersville, Pa., to Drexel Hill, Pa., 461 Harper Ave.

Whiton, Ross K., from Bedford (Middlesex North) to Concord (Middlesex South), 7 Sudbury Rd.

Wilson, Charles O., Lowell, from 175 Fairmount St. to 614 Gorham St.

Wood, Benjamin E., Boston, from 520 Beacon St. to 475 Commonwealth Ave.

1924 *Wood, Gilbert Osborne*, died at Framingham, Jan. 19, 1925, aged 47.

Yudin, Hyman, from Salem to Beverly, 497 Rantoul St.

Boston Medical Library

Recent Accessions

Babcock, R. H. Your heart and how to take care of it. N. Y., Appleton, 1924.

Bacmeister A., and Rickmann, L. Die Röntgenbehandluug der Lungen- and Kehlkopf-tuberkulose. Lpz., Thieme, 1924.

Baldwin, J. C. Pediatrics for nurses. N. Y., Appleton, 1924.

Bowers, P. E. Manual of psychiatry. Phila., Saunders (c 1924).

Braun, W., and Wortmann, W. Der Darmverschluss und die sonstigen Wegstörungen des Darmes. Berl., Springer, 1924.

Borries, G. V. T. Zur Klinik des Nystagmus. Berl., Urban, 1924.

Bridgman, G. B. Bridgman's life drawing. N. Y. (c 1924).

Brown, W. L. The sympathetic nervous system in disease. 2 ed. Lond., Prowde (1923).

Brandeis, J. W. The extraordinary exploits and experiences of Munchausen, M.D. N. Y., 1924.

Bürger, M. Pathologisch-physiologische Propädeutik. Berl., Springer, 1924.

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Resignation of Staff of Malden Hospital

DOCTORS W. S. Prior, R. W. French, F. W. Gay, F. A. Hodgdon, C. C. Burpee and J. J. Corbett, constituting the Staff of the Malden Hospital, have resigned because the recommendations of the Staff have been ignored by the Executive Committee of the Hospital. The physicians will continue to care for patients in the Hospital. We are informed that Mrs. Alice G. Burnham, a member of the Executive Committee and also of the Board of Trustees of the Hospital, has also resigned without giving any reason therefor.

Scarlet Fever Antitoxin

Through the interest of His Honor, Mayor James M. Curley, the Trustees of the Boston City Hospital have been able to secure a supply of scarlet fever antitoxin for use in the South Department, Boston City Hospital.

It was planned to have a supply adequate not only for use in the hospital but for physicians who might need the antitoxin for their patients.

This supply is now available and will be supplied to physicians at cost.

Application may be made to So. Dept. in person, by letter, telegram or telephone.

RECENT DEATHS

FARNHAM.—Dr. Charles Chittenden Farnham, president emeritus and former secretary of the College of Physicians and Surgeons, Boston, died at his home in Randolph, Mass., March 8, 1925, at the age of 86.

He was born at Clinton, Conn., was educated at the University of Rochester and at the College of Physicians and Surgeons, Boston, where he received an M.D. in 1882. He was active in the affairs of the First Baptist Church and a former superintendent of its Sunday school. He abandoned his practice in Randolph in 1917, at which time he also retired from his post as town librarian, a position he held for 41 years. In 1877-78-79 he was town clerk and treasurer. In 1874 he married Anne T. Howard of Randolph. He is survived by a daughter, Mrs. Walter F. Stephens, two grandchildren and two sisters.

"The Lord giveth and the Lord taketh away. Blessed be the name of the Lord." Facts from "Quarterly Announcement" of Coll. P. and S., Boston, 1924.

KENNARD.—Dr. Harry Delano Kennard, a Fellow of the Massachusetts Medical Society, died of heart disease at his home in Peabody, March 6, 1925, at the age of 56 years.

He was born in Beverly, was a graduate of Harvard Medical School in 1895, and was a member of the Peabody Board of Health and of the staff of the J. B. Thomas Hospital. He is survived by his widow and two sons.

OBITUARY

Resolutions Adopted by the Somerville Medical Society on the Death of Dr. Charles F. Maguire

Whereas: Almighty God, in His Divine Omnipotence, has been pleased to remove from our midst in the field of medicine, our colleague and co-worker,

DR. CHARLES F. MAGUIRE,

Be it

Resolved: That we, members of the Somerville Medical Society assembled, deeply mourn his great and irretrievable loss.

An upright man; an able and sympathetic physician; a student, distinguished in college, in the professional schools, and through life for his scholarly attainments; a genial friend to all; a citizen whose public and private acts were above reproach; a father and husband loving and devoted to his family:

Eminently proficient and extremely modest in the practise of medicine, cheery and helpful to all who sought his aid, we feel that the community and the medical profession have met with a great loss.

Be it further

Resolved: That these resolutions be spread on the records of the Somerville Medical Society, and copies be sent to the bereaved family, to the Boston Medical and Surgical Journal, to the *Journal of the American Medical Association*, and to the press.

Michael W. White,
Giles W. Bryant,
Edmund H. Robbins.

CORRESPONDENCE

CHARLES FRANCIS MAGUIRE

Mr. Editor:

The passing of a man of the character of Dr. Maguire deserves more attention than is accorded by the conventional obituary notice. In his chosen sphere of general practice he was alert, splendidly balanced and most resourceful. Better still, he was honesty and kindliness personified. By the people of Somerville, among whom he labored, he was worshipped. The untimely death of such a physician is a real calamity.

J. W. Courtney.

Boston.

LEPROSY WORK AT CULION

Office of the Governor-General
of the Philippine Islands
Manila, February 2, 1925.

My dear Sir:

I am sending you a copy of certain very interesting reports with reference to leprosy work at Culion. We are having an uphill fight to interest people in this great effort. I am sending you this for your information and hope it will be of such interest that you will give it prominent reference in your journal. Such action on your part will help very much in interesting the medical profession and eventually the American public.

Very respectfully,
Leonard Wood,
Governor-General.

The Editor,
Boston Medical and Surgical Journal,
Boston, Mass.

THE BRITISH EMPIRE LEPROSY RELIEF ASSOCIATION

24 Cavendish Square, London, W. 1.
October 21, 1924.

H. E. Governor-General Wood,
Manila.

Your Excellency:

It gives me great pleasure to accede to your request that I should place on record my impressions of my recent visit to the Culion Leper Settlement, and before I do so I should like to take the opportunity of very warmly thanking you for your great kindness to me while I was in the Philippine Islands. I wish, also, to record my indebtedness to your personal staff, the officers of the Health Bureau, as well as all of those in charge at the Settlement itself. One and all did their best to help me make the enquiries I had in hand and their help was invaluable. I am very greatly in the debt of all with whom I have had anything to do, and the information I have been able to obtain, accompanied as it is by the splendid series of photographs which I received at Culion, will be of great service to me in all my future work among the lepers of our British Empire. I warmly thank you.

Your Excellency has asked me to record my opinions on the work being done at Culion and I will gladly do so if it will be any help to those responsible for what is being done.

First of all I wish to say that I believe that the policy of segregation is the best policy for a country which has, as you have in the Philippine Islands, a limited number of lepers. As far as the Philippines are concerned I am inclined to believe that the policy that has been pursued since 1906 has resulted in a reduction in the number of lepers in the Islands. Dr. E. Muir, in his "Handbook on Leprosy," page 82, quotes an article on Culion, I think it was written by Dr. Victor Heiser, as follows: "The present status

of the problem is in striking contrast with that of 1906, when lepers were encountered almost everywhere without any restrictions. In a number of instances they worked in cheese factories, as salesmen in grocery stores, as coachmen, school teachers, clerks, in tobacco factories, and at other similar pursuits."

Now, Your Excellency, I have made numerous enquiries since I have been in Manila and I am informed that there is hardly a leper to be seen in a town of any size, in public, while there are comparatively a small number of advanced cases in Culion, and hardly any at the San Lazaro Hospital. This seems to indicate that the old advanced cases have died off while the disease is being controlled in many of the cases now segregated. In addition to this it is undoubtedly true that many early cases are coming forward for treatment, at San Lazaro in particular and some at Culion. Both these facts point in the direction of there being some reduction in the numbers of lepers in the Islands. I suggest, therefore, that the policy of segregation has not been altogether a failure, by any means, as suggested by some critics; indeed, I think it has been more successful than some realise.

If there were to be plenty of money available for leper work, that is, much more than has been available so far, it might be considered advisable, as is the opinion of the Culion Medical Board, with which opinion I am in agreement, that local treatment stations might be commenced at provincial centers. If, however, as seems to be most likely, no more money will be available, then I strongly feel that all the money there is should be spent on Culion. If these treatment stations are commenced now it must mean that funds now spent at Culion will be taken for the new work and so the work at Culion will suffer. Nor is there the personnel, as far as doctors are concerned (so I am informed), for these new stations without taking men from Culion, where more men are needed for the treatment, not less. With a stationary appropriation for leper work Culion should come first. It is better to make the one center first class than to have several places, none of which is in any way what it should be. My judgment is, therefore, that till much more money is available there should not be the opening of any number of treatment stations. If funds, apart from those now needed for Culion, can be obtained, then, I think, it might be wise to have two or three local treatment stations for early cases.

I consider that the administration of the Settlement is very good. The officers in charge are keen and competent, they are alive to the needs of those in their charge, and aware of the improvements that ought to be made, but which are impossible, or seemingly so, because "there is no money." If only more money were available a great deal could be done to make the whole work of the Settlement more effective.

The actual working arrangements are, on the whole, to be commended. The housing is pretty good, though not entirely adequate to the needs of the population. Food is, generally speaking, suitable, although improvements, as suggested by Dr. Miss Embry, might still be made if funds were more plentiful.

There is a general air of well-being that immediately strikes a visitor. The lepers appear to be contented. I spoke to three of the American lepers and all said that things were about as good as they could be. They spoke well of the administration and general management and appeared to be quite satisfied with existing conditions. A few are sure to be discontented, but the majority are satisfied.

The medical staff is excellent. Dr. Wade and Dr. Perkins are men of international repute in circles where leper work is considered, and they and their colleagues are keen and alert. They are, indeed, doing work which is proving useful to leper workers all over the world. More doctors are needed for giving the treatments, as well as for research work, and better equipment is needed in some directions if the best work is to be done.

The education given in the schools seems to be what is needed and the children whom I saw in school seemed bright and happy.

Taking the Settlement as a whole, I must confess that I was pleasantly surprised to find it so well laid out and so well equipped. The theater, reading room, the provision of bands, to mention two or three things only, clearly showed that there has been untiring energy expended in providing for the comfort and the happiness of the unfortunate lepers.

Culion stands by itself in the whole world. It is the largest Settlement for lepers and, as far as I am aware, the best. The government of the Philippine Islands and the people ought to be proud of Culion and eager to keep it up to its present standard, and, where it is possible, improve it. I believe it can be improved and so do those on the spot. Culion for many years has stood as the outstanding example of what can be done and has been an inspiration to many workers among lepers in other lands.

In a word, I consider Culion to be a splendid institution and I would strongly urge that a publicity campaign should be undertaken by government and by the press with a view to counteracting misstatements that are apparently being made, and with a view to letting the people know what Culion is really like. The chief of the colony suggests that cinematograph pictures of life at Culion should be taken and exhibited in different centers in the Lslands. I think the suggestion an excellent one. When I had the pleasure of meeting the newspaper editors on Friday last I appealed to them to make it known everywhere that Culion was a splendid place, and I hope they will do something in the matter.

The main point to be stressed to the public is that Culion need not necessarily be looked upon as a place where lepers are to be permanently segregated, but as a place to which lepers may go for a period of treatment with a real hope of recovering. That the very best doctors are available there and that the latest and most improved treatments are being used, with the most wonderful results. I think if it were widely known that already 104 lepers have been sent away as "cured" between January and the beginning of October this year, and that, in addition, there are 222 lepers who are negative and who are undergoing the two years' observation period, a different view would be taken of the worth of Culion.

Of course the very success of the treatment will bring other problems which must be faced. One of these is the need of provision of plots of land, and perhaps houses and some maintenance, for lepers who have recovered and who have no homes, or friends, to whom they can go. Apparently a good number, even if they get better, do not wish to leave Culion, and I understand that more than one who has been sent away as cured has asked to be allowed to return and live at Culion. There seems to be plenty of land which might be used for this purpose. I think this should be looked into.

Immediate attention ought to be given to the question of communication with Culion. I am informed that the conditions of transport (for lepers) leave much to be desired, and that because of this many lepers remain hidden and refuse to come to Culion. Men, women and children, educated and uneducated, well-to-do and the very poor, are herded together on the steamer, and this ought not to be so. Then, also, lepers would probably be more willing to go if they did not feel so completely cut off from their friends and relatives. Moreover, improved communication would enable foodstuffs to be more easily transported than at present, and it seems that at times there is

a shortage of some articles of diet because no steamer is available.

Dr. McKean of Siam, in his letter to you after his visit to Culion, dealt with the question of the lepers having some work to occupy their attention, and I warmly endorse his suggestions, and I am glad to note that so many of the lepers are engaged in fishing and gardening and other occupations.

In my judgment there is one matter of outstanding, and very grave, importance that ought to receive prompt attention and action. I refer to the leper and untainted children.

I must say I was horrified to learn that there are some 1300 leper children in the Settlement (the figures were supplied to me by the chief of the colony). Such a large proportion of child lepers is, I venture to think, unusual. In India, for instance, taking the figures for the 62 leper asylums which are connected in one way and another with the Mission to Lepers, there are the following (and I compare them with Culion figures):

	Indian asylums	Culion
Male lepers	4300	2700
Female lepers	2750	1400
Adults	7050	4100
Child lepers	400	1300
Untainted children	700	250

This matter ought to be dealt with at once. I know well that the matter has been carefully considered by you, but there ought to be no delay in making some arrangement for these children.

In the first place, the leper children ought to be living in special dormitories or hostels by themselves. I believe that some of them are doing so. The system ought to be extended to all. The early cases ought to be kept in observation hostels where they do not come into contact with advanced cases. Every leper child ought to be receiving the latest treatment, unless the doctors decide that for other reasons this should not be given. If the large majority of the child lepers were kept by themselves away from the worst cases and were treated, I believe, following out experience in India, many of them would become negative in a short time.

Even more important than this, if it can possibly be so, is the question of the 250 (the number given me) of untainted children now at Culion. Of these healthy children not more than 40 are in the special Home for Untainted Children in Balala. The other 200 healthy children are apparently still living, either with their leper parents or with the leper children. We all know that children are specially susceptible to infection, and it is a very great pity, not to put it more strongly, that these healthy children should be compelled (and I think that this is the correct word to use) to live with lepers in the Settlement, where many of them will most likely contract the disease.

I do not shut my eyes to the difficulties by which you have been faced, but I do feel that you will not mind my stressing in this way the importance of something being done for these children.

All untainted children ought to be kept in special homes (like the existing one at Balala), and I suggest that these children ought to be kept at Culion till they reach the age of 10 or 12. If they are all forcibly removed to Manila, as I believe has been proposed, discontent among the parents will be caused which will adversely affect public opinion toward Culion, and the children will be homesick and restless; if kept at Culion the children will be near the best medical opinion and attention available anywhere in the Islands, and, if the disease shows itself, they can be treated immediately and cured; while, further, if they are all removed to Manila and after a few months there the disease shows itself in some of them, as is likely to happen, it might cause a small panic and do serious damage to the existing orphanage work. If they remain at Culion they are in a climate to which they are used and they can see their parents or relatives occasionally. It would, moreover, probably be more economical to keep them at Culion while they are young and then, when they are 10 or 12, they might be drafted to a special home in, or near, Manila where they could receive further education with the orphanage children, or be trained for some work.

This work of providing for the untainted and leper children ought to be taken in hand at once and as many of the little ones as possible saved from becoming lepers. Some six new homes ought to be planned for the untainted children and at least the same number for the leper children who are early cases.

If the Philippine government is unable to find the funds for this most necessary piece of work I feel sure that the American public would willingly find what is necessary if an appeal were made to them for this specific purpose. A certain number of hostels could be asked for, to be named after donors, and a sum of money to equip and endow them, so that the Philippine government would be freed from the fear of having to find money for this special piece of work in the future. I venture to say that nothing would appeal to the public as an appeal for such work, and I write after considerable experience in the matter.

I trust, Your Excellency, you will not feel that I have been too free in writing what I think about the work at Culion. I am filled with admiration for it as a whole and I do congratulate you on the improvements which I am told have been brought into being during your term of office. It is a wonderful piece of constructive work and my only desire is that it should be even better than it is, and more effective, as I believe it may be.

With warm thanks for the opportunities you gave me for consultation with the workers in Manila and at Culion, and assuring you that if there is any matter in which I personally can be of service to you you have only to command me,

I have the honour to be,
Your Excellency,
Your obedient servant,
(Sgd.) FRANK OLDRIEVE,
Secretary.

REMARKS ON CULION LEPER COLONY

BY DR. A. R. J. DOUGLAS, RANGOON, BURMAH

Armed with a letter from the Philippine Health Bureau authorizing my visit to Culion and asking the authorities there to facilitate my inquiries, I have spent nearly a fortnight at the leper colony. During this time I have had frequent conversations with Dr. Jose Avellana Basa, chief of the colony; Dr. Wade, the chief pathologist, and Dr. Lara, the chief physician, to all of whom I am greatly indebted for the trouble they have taken in placing all available literature and figures at my disposal and in helping me to arrive at accurate conclusions with regard to the questions of segregation as applied to Culion and the modern treatment of leprosy as carried out there. As I have lived with the medical staff there I have had also unusual opportunities for ascertaining their views, which have been very helpful to me. The medical members of the staff have spared no pains to show me their cases and to discuss the varying conditions shown by the patients. I would especially mention Dr. Nicolas, who, among other things, showed me the children. Last, but not least, I mention the chemical department under Dr. Per-

kins, the chief chemist, who very kindly showed me the work of manufacturing the medicines in the factory and explained the general lines along which he was searching for new and improved remedies.

I do not think outside of those officials directly concerned that the magnitude and value of the work being carried out at Culion is realized. There is nothing like it in the world, either in India or elsewhere, as far as I am aware. Nearly 4000 patients are undergoing the different forms of present modern injection treatment. They are taking this treatment voluntarily and hopefully, and, speaking very roughly, one in three shows before long definite signs of improvement, not merely visible to the doctors but also sufficiently marked to be seen by the patients themselves. This work on the present scale has been going on now for about two years, but before definite conclusions can be drawn another two or three years will be required, and, in my opinion, any alteration of policy which would in any way limit or check this work would be a most grievous misfortune, not only for the lepers in the Philippines but for those unfortunate people all over the world.

The work being done and the self-sacrificing expenditure will stand as a monument to the Philippines for all time in the history of medicine.

I understand that there is a considerable body of opinion in favor of reducing the number of lepers at Culion and sending all early cases of leprosy to local treatment stations at Cebu and other centers in the Islands. The idea being that if patients can be treated at centers near their homes they will come forward voluntarily in greater numbers and there will be fewer cases of concealment of the diseases owing to horror of going to Culion. Theoretically I agree that the modern treatment can possibly be carried out more accurately in such treatment stations, but I am strongly of opinion that the results actually obtained would in no way correspond to those theoretically expected.

The work in different institutions hundreds of miles apart must vary, and the statistics would be open to so many outside factors that the scientific value of the work not only would be much less, but the whole system of segregation and the modern treatment of leprosy might crumble away like a house of cards.

In conclusion, I may repeat that I am strongly of opinion that the present system which has gone on for 17 years should be allowed to continue for another three years and then a stage will be reached when work on different lines can be started if considered desirable. The whole question of segregation is greatly changed by the modern treatment, and if this is successful it may cease to be the apple of discord.

Hongkong, 4th February, 1924.

Hon. Eugene A. Gilmore,
Lieutenant-Governor of the Philippines,
Manila, P. I.

Dear Governor Gilmore:

May I ask that you will not consider it presumption for me to hand you herewith a memorandum of some of the items which I have mentioned verbally to you and to Dr. Avellana Basa and to others who are deeply interested in the welfare of Culion Leper Colony?

I refer particularly to possible measures or methods of production in the colony that would not only give interesting and profitable employment to such of the leper population as are able for it but also would be a source of income, thus lessening in a degree the annual outlay of government funds for maintenance.

May I mention first the planting of crop-producing trees?

The Rain Tree. I am unable to give the botanical name of this tree. It commonly is called the rain tree and grows freely in Manila and in Culion.

This tree will produce sticklac when properly infected with the sticklac producing insect (whose proper name I have forgotten also). This tree grows also in Siam. It is easily planted, grows rapidly, and in eight years from the planting of the seed may be used to produce sticklac.

In the Chiengmai Leper Asylum during 1922 we gathered from our few adult rain trees sticklac that sold for over 5000 rupees (Rs. 5428), the equivalent of about $1600 gold. There are some 2000 rain trees on the asylum grounds and we expect to derive a considerable income from them when they have reached the proper age.

Whether the sticklac producing insect will thrive in the Philippines I do not know. But it certainly is worth the trial. When our next crop matures (December, 1924) I shall be very glad to send a supply of the embryo insects for a trial. These might be tried out on some of the full-grown trees in Culion. In case it thrives all those mountain sides near the colony might be planted out to rain trees. They require but little care. Twenty trees when full grown should produce sufficient sticklac to support a leper family. There is an increasing demand for sticklac in the United States and the present price is 25 cents gold per pound.

Kapok (Tree Cotton). This tree is planted with great ease, thrives on Culion and could be planted broadcast. There is a growing demand for this tree cotton in the U. S. A., the price ranging from 17 to 34 cents gold per pound.

Acacia Catechu. I do not see this tree in Culion or elsewhere in the Philippines. But as it grows in great abundance in Siam I presume it will do well here. Once planted it requires no care and is self-propagating. The heart of the tree and its roots are chipped and boiled for the extraction of catechu, which always is in demand at a good price.

The wood is hard and very durable, and valuable for house posts and similar uses.

Pili. I know this tree by reputation only. Professor Higgins of the Agricultural College tells me that it is slow in coming into bearing. Its nuts are so valuable, however, with a constantly increasing demand for them in the U. S. A., that it would seem to be a most valuable tree for the Culion hills.

Cocoanuts. It goes without saying that cocoanut planting, well done, is profitable. Whether it would be profitable in Culion is a question inasmuch as it might not be possible for leper people to give the care, fertilization and other attention which is necessary to produce a paying crop.

Cattle Raising. On the return journey from Culion I had as a fellow passenger an American planter from Buswanga by the name of Parmelee. He told me that he supplies Culion Leper Colony with fresh beef to the extent of from 1000 to 1200 beeves per year. I understand that a large proportion of these are produced on his ranch in Buswanga.

Would it not be feasible to do cattle raising on Culion? I presume that the conditions there are equally good with those in Buswanga nearby.

No doubt the Filipino people are adept in raising domestic animals and that a leper family supplied with a pair of breeding cattle would in a few years be possessed of a herd, the increase of which would go far toward their support from the sale of beef and the production of milk for the family.

Carabao. Parmelee says he raises carabao also on his ranch and that there is a constant demand for them, and that the average price of a work carabao is 100 pesos.

Goats. Possibly the raising of goats would not be practicable, but with the very wide range for grazing on Culion it would appear to be an ideal place for raising this hardy animal. The goat multiplies rapidly and herds of goats raised by leper families

would help to solve the meat supply of the colony and provide milk for the family also—a matter of real importance to those ill with leprosy.

Chickens. As the chickens and eggs used in Culion must come from Manila it would seem that the raising of these fowls by the leper people would be very useful and profitable as well. I do not know that suitable food for chickens could be produced there, but if it could chicken raising probably would be entirely feasible.

Inasmuch as I am not familiar with all of the conditions in Culion some of the above suggestions may be wide of the mark, but I offer them for whatever they may be worth.

There is one problem in Culion in connection with the successful treatment of leprosy, namely, the matter of provision for the increasing number of negative cases, eligible for parole.

Colonel Munson tells me that Governor-General Wood has been trying, without success, to secure employment for a paroled man formerly a leper. No one cares to employ him. People are afraid of him. Although cured, or at least no longer a menace to anyone, he still carries the stigma of once having been a leper.

What is to be done with the more than 200 negative cases now on the list?

Would it be practicable to arrange for a negative colony on some fertile area of the island and supply these negative people with the beginnings of herds of cattle, carabaos, goats, chickens, pigs, etc., in order that they may become self-supporting and also furnish a source of supply to the leper portion of the colony?

Perhaps some such plan already is in the minds of those having the work in charge.

It must go without saying, however, that to ensure success in the lines of work suggested above it would be necessary to put them in charge of men who not only know conditions in Culion and who understand the mind and temper of the Philippine people, but also and particularly men with real heart and of a high purpose and with a real desire to bring the highest good to the unfortunate leper people.

Industrial Training. As to the subject of industrial training I need only refer to it, as you already are deeply interested in the matter and have made a good beginning in the establishment of the laundry and the tailor shop.

Although I did not get to visit the public school in Culion, I was gratified to know that there were nearly 300 pupils in attendance.

If an industrial department, along simple lines at first, could be operated in connection with the public school, supported by school funds, it would be a great boon to the children of Culion, of whom there are 500 under 18 years of age and a total of 1000 or more under legal age.

Handicraft is being taught in the public schools in the province, and I was astonished at the products turned out by the pupils (which Mrs. Gilmore very kindly showed me at the Educational Bureau salesroom).

If the more homely and practical crafts were taught, such as carpentry, basketry, cabinet making, blacksmithing, pottery, and possibly also weaving, lace making, embroidery, etc., it is possible that a considerable portion of the output would be used in Culion town of almost 6000 persons, to the mutual advantage of both buyer and seller.

Even though there were no outside demand for Culion products yet the training given the young would well be worth the cost on account of the large moral effect; for experience shows that one of the vexatious problems of colony or asylum life is that of a method of occupying the time and energy of the young and those who have the ability and the desire to work.

My visit to Culion was most interesting and instructive and I thank you heartily for the arrangement you kindly made for me there.

I am sincerely glad that the affairs of this great leper colony are under your guidance and that you have the hearty coöperation and support of Governor-General Wood and the interest and good will of all right-minded people.

Yours very sincerely,
(Sgd.) JAMES MCKEAN.

Peking Union Medical College
Peking, China
Department of Pathology

September 19, 1924.

General Leonard Wood,
Governor General,
Philippine Islands.

Dear Sir:

Through the courtesy of Dr. Vicente de Jesus, Director of Public Health, Philippine Health Service, I had the opportunity recently to visit the Culion Leper Colony. The trip proved interesting and instructive in the greatest degree, largely because of the fact that the work being done there was so very much worth while. No one could fail to be interested. It is apparent that the authorities have felt that this work was one of the most important being carried on, for the benefit and happiness of the Philippines, and it should be so considered by all who are in the least loyal to their government, or who have hopes of developing greater things for their country.

In the first place, the large number of patients on the island impresses one. Just what proportion of the entire lepra population of the islands this is I do not think anyone can say, but it must be a large part of it. The second thing that is noticeable is the physical comforts that are available for the patients. Their houses compare favorably with those seen in the native portions of Manila, and, taken as a whole, the living conditions are certainly infinitely more satisfactory than in the dungeons the lepers are kept in at San Lazaro Hospital. In many ways the inmates are undoubtedly far better off than before they were taken to Culion or than they would be if they were released. I heard, nor saw, no evidence of dissatisfaction on the part of the patients. The very fact that the colony contains so many patients means that its successful administration is a big job, and that its future success depends largely upon the vision of those responsible for the health of the people.

In one of our conversations you mentioned the fact that you had plans and hopes for wider development on the island, thus making the lot of these people better and the hastening of the completion of the problem of the eradication of leprosy in the islands. I hope that you will soon be able to put these plans into operation. I have thought several times about the drive to raise money for the Rizal Memorial. I hope they get the money, but I have wondered if it would be possible to raise as much to put into a work which would probably stamp out leprosy in one generation and add another heritage of physical cleanliness to the members of the next generation.

The food furnished is apparently sufficient. There are only two criticisms that I can make in this connection. First, insufficient variety. This would automatically be remedied if by the development of a system of good communication through the island the people could get onto the soil and, by growing food, help themselves and the others, not able to work. Secondly, the lack of protection of the food supplies from flies. This is, of course, a local sanitary measure and should be remedied by those in present authority on the island.

The fresh water supply is apparently not quite adequate, but steps are being taken to increase it. Two

artesian wells drilled in the early days of the colony have been equipped with pumps, but this source seems unsatisfactory at the height of the dry season, while at other seasons an abundant supply can be had from the stream. Progress on this work seems astonishingly slow. Here, as everywhere in the Philippines, water enters as a very prominent problem in public health administration.

The statistics of the time since ample funds were placed at the disposal of the staff of Culion to carry on adequate treatment show that, of the patients treated, 56 per cent. are improved and 36 per cent. are stationary. The figures also show that the majority of deaths at Culion are not from leprosy but from some intercurrent disease, usually many times this disease being tuberculosis or nephritis. These facts are interesting if read together. It means that segregation and systematic treatment are solving one problem, namely, it is controlling leprosy. But it means this also, that with a hospital population of over 5000 patients, the general clinical care received is inadequate and arrangements should be made to place a larger number of competent clinicians on the field there. It means that leprosy undermines and the other disease kills. This is not peculiar to leprosy, nor is it peculiar to Culion; it is probably the same condition that portends in the many still unsegregated cases in the islands. It means that by tightening up on the general clinical work done the percentage of those cured would be greatly increased. I wish to state emphatically that this is not in any possible way an argument against segregation, but one for more adequate segregation, and, above all, earlier segregation.

Conditions as they exist in the Philippines offer an outstanding opportunity for the possible solution of an age-old question in the epidemiology of leprosy. It is known that a majority of the patients segregated at Culion come from one island. This offers an opportunity, that should not be disregarded, to make a concerted effort to solve the problem of the actual method of transmission of leprosy.

After my return to Manila I read some interesting press notices, and heard still more astonishing remarks, about the abolition of segregation for lepers in the Philippines. It is unnecessary to go into each one as they can all be answered in the same way. One of the chief arguments seems to be that, in view of the fact that leprosy is one of the least contagious diseases, segregation should be abolished. Although one of the least contagious, it is one of the most intractable of the chronic infections and therefore should be isolated. The fact that tuberculous patients are not segregated is no argument. All mankind is susceptible to myriads of infectious diseases, and if we extended the same argument against segregation of such diseases down the list we would abolish at one blow the backbone of modern preventative medicine. We do know that by segregation, combined with treatment, we can control and eradicate leprosy, and it should therefore be carried out.

To abolish the efforts for leprosy eradication in the Philippine Islands during the last generation by turning all the patients loose would be about as sensible as closing our hearts, minds and books and vaulting back into the medical dark ages, and wish the rat, the mosquito, the flea and fly long life and bon voyage because we have no moral right to curtail their normal activities. Segregation, even if discontinued officially, would continue, for the people actually segregate their lepers, usually under conditions vastly worse for the lepers than exist at Culion or even at San Lazaro.

History has shown that segregation has been the only answer in the past—it is certainly more than half the answer today, and should be accepted as a fact and not as a plaything for near-sighted politicians, whose real object in life is votes and not public good.

Instead of fighting against segregation every really public-spirited citizen should be working to make segregation more effective, and if they feel the impulse to make the lot of these unfortunates lighter there is certainly much money needed for further work in leprosy prevention and it should be found, through some channel.

Sincerely yours,

C. L. BARTLETT, M.D.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

DISEASES REPORTED FOR THE WEEK ENDING JANUARY 24, 1925

Diseases	*No. of Cases*	*Diseases*	*No. of Cases*
Anterior poliomyelitis	3	Ophthalmia neonatorum	35
Chickenpox	283	Pneumonia, lobar	158
Diphtheria	107	Scarlet fever	388
Dog-bite requiring anti-rabic treatment	4	Septic sore throat	4
Encephalitis lethargica	8	Syphilis	47
Epidemic cerebrospinal meningitis	2	Suppurative conjunctivitis	15
German measles	161	Trachoma	2
Gonorrhea	88	Trichinosis	4
Hookworm	1	Tuberculosis, pulmonary	135
Influenza	124	Tuberculosis, other forms	19
Measles	380	Hilum tuberculosis	1
Mumps	102	Typhoid	8
		Whooping cough	140

DISEASES REPORTED FOR THE WEEK ENDING FEBRUARY 7, 1925

Diseases	*No. of Cases*	*Diseases*	*No. of Cases*
Actinomycosis	1	Pneumonia, lobar	178
Chickenpox	239	Scarlet fever	349
Diphtheria	106	Septic sore throat	6
Encephalitis lethargica	3	Syphilis	49
Epidemic cerebrospinal meningitis	2	Suppurative conjunctivitis	16
German measles	368	Trichinosis	3
Gonorrhea	80	Tuberculosis, pulmonary	122
Influenza	62	Tuberculosis, other forms	20
Measles	429	Typhoid fever	10
Mumps	118	Whooping cough	119
Ophthalmia neonatorum	28		

DISEASES REPORTED FOR THE WEEK ENDING FEBRUARY 14, 1925

Diseases	*No. of Cases*	*Diseases*	*No. of Cases*
Actinomycosis	1	Ophthalmia neonatorum	40
Anterior poliomyelitis	1	Pneumonia, lobar	243
Chickenpox	247	Scarlet fever	410
Diphtheria	136	Septic sore throat	5
Dog-bite	4	Syphilis	56
Encephalitis lethargica	3	Suppurative conjunctivitis	30
Epidemic cerebrospinal meningitis	3	Tetanus	1
German measles	538	Trichinosis	1
Gonorrhea	105	Tuberculosis, pulmonary	127
Influenza	89	Tuberculosis, other forms	31
Measles	572	Typhoid fever	14
Mumps	92	Whooping cough	170

NEWS ITEMS

Restoration of Registration of Dr. Thomas W. Leavitt

AT a meeting of the Board of Registration in Medicine, held February 26, 1925, the Board rescinded its vote of February 12, 1925, whereby the registration of Dr. Thomas W. Leavitt of 338 Ferry St., Malden, Mass., was suspended for six months, and the case placed on file.

Joins Health Board

Ware, March 10—Dr. Harry D. Gafney has been chosen a member of the board of health. Dr. Gafney is also a member of the school board.

Dr. W. W. Keen Honored

IT is reported in *Science* that Dr. W. W. Keen, of Philadelphia, has been elected a foreign honorary member of the Royal Academy of Medicine of Belgium.

Dinner to Dr. Ross V. Patterson

During the visit of Dr. Ross V. Patterson, Dean of the Jefferson Medical College of Philadelphia, a dinner was tendered him by a delegation of the local alumni at the Copley Plaza Hotel, as representative to the meeting of members of the Association of American Medical Colleges.

He gave a brief address on the growth of the College, citing its prosperous condition and describing its new hospital, laying stress on the Centennial Celebration to be held the latter part of May after the American Medical Association meeting at Atlantic City.

The dinner was in charge of Dr. Wallace P. MacCallum, Alumni Representative for Massachusetts, and was attended by Drs. Joseph Fallon of Brookline, Charles A. Riley, Brookline; Gustave Hartman, Lynn; James A. Mansfield, Dorchester; Edward R. Fleming, Medford; P. F. Gahan, Medford; E. W. Barrett, Medford; H. J. Walcott, Concord; Henry Hirsch, Boston; Wallace P. MacCallum, Boston; L. W. Harris, Cliftondale; Frederick C. Hemeon, Dorchester, and John S. May of Roxbury.

REPORTS AND NOTICES OF MEETINGS

Notices of meetings must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.

A PHYSIOLOGICAL CONFERENCE will be held Wednesday, March 25, in the Bowditch Library, Building C, Harvard Medical School, at 4 P. M. Dr. Alexander Forbes will speak on "The Frequency of Motor Nerve Impulses in Sustained Reflex Contraction of Skeletal Muscles."

Harvard Medical Society

THE next regular meeting of the Harvard Medical Society will be held as usual in the amphitheatre of the Peter Bent Brigham Hospital, Mar. 24, 1925, at 8:15 P. M. The program follows:

1. Demonstration of Cases.
2. Cystological Classification of Gliomata and its Prognostic Significance—Drs. Percival Bailey and Harvey Cushing.

All members of the Medical Profession, Medical Students and Nurses are invited.

The Massachusetts Psychiatric Society

A MEETING of the Massachusetts Psychiatric Society will be held at the Boston Psychopathic Hospital, 74 Fenwood Road, Boston, Mass., Tuesday, March 31st, at 8 P. M.

PROGRAM

Dr. William T. Hanson—"Problems Concerning the Criminal Insane"
Mr. Herbert C. Parsons—"Juvenile Delinquency and Probation"

R. M. CHAMBERS, M.D.,
Secretary and Treasurer,
Mass. Psychiatric Society.

Health Unit, 17 Blossom St., Boston

THE monthly meeting of the West End Neighborhood Conference will be held at the Health Unit, 17 Blossom St., Friday, March 20, at 3.30 P. M. Dr. Percy R. Howe, who has been conducting some extremely interesting experiments, as well as contributing valuable information in the field of dental hygiene, will speak on "Relationship of Diet to Teeth." Dr. Howe is on the regular list of speakers in the Harvard course of lectures open to the general public and his talks have created a profound impression and attracted large audiences. The members of the West End Neighborhood Conference ought to be congratulated in this presentable opportunity to have Dr. Howe with us.

Please manifest your interest not only in the monthly conferences conducted at the Unit but in the important subjects which Dr. Howe will discuss.

Please be sure to be present or send someone to represent your agency.

CHARLES S. WILINSKY, *Secretary,*
West End Neighborhood Conference.

The Boston Dispensary, 25 Bennet Street

THE Staff of the Boston Dispensary is to hold during the current year several meetings devoted to the general discussion of dispensary and out-patient work. The first of these meetings is to be held March 26th, at 12:30 P. M.,

to be addressed by Dr. Roger I. Lee on the subject: "The function of the Out-Patient Department. From the standpoint of opportunity in preventive medicine, early diagnosis and health education."

Anyone desirous of attending this meeting will be invited on application to the Secretary of the Medical Staff.

A. K. PAINE, M.D.,
President.
BENJAMIN E. WOOD, M.D.,
Secretary.

Meeting at Harvard Medical School

ON Monday afternoon, March 2nd, Dr. George B. Magrath, Medical Examiner of Suffolk County, addressed a gathering of the students at Harvard Medical School, on "Sketches from the Log-book of a Medical Examiner." Dr. Magrath told of some interesting investigations he had conducted in connection with his duties and illustrated them with lantern slides.

The meeting was held under the auspices of the Medical School Committee of the Phillips Brooks House Association.

Association of American Medical Colleges

THE following officers were elected at the annual meeting of the Association of American Medical Colleges to serve for the coming year:

President—Hugh Cabot, M.D., Dean of University of Michigan Medical School.

Vice-President—David L. Edsall, M.D., Dean of Harvard Medical School.

Secretary and Treasurer—Fred C. Zapffe, M.D., of Chicago, Ill.

Executive Committee—Dr. Walter L. Niles of Cornell, Dr. Irving S. Cutter of the University of Nebraska, Dr. Charles P. Emerson of Indiana, Dr. Ray Lyman Wilbur, President of Stanford University, Dr. Zapffe and Dr. Charles F. Martin of McGill University.

The annual meeting will be held the last week in October hereafter, instead of in March. The next meeting will be at Charleston, S. C.

The Lawrence Medical Club

THE Monthly Meeting of the Club was held Monday evening, Feb. 23, with J. Forrest Burnham, M.D., Red Tavern, Methuen.

Chairman for the evening was Henry F. Dearborn, M.D. Subject: Surgical Diseases in Children, by George D. Cutler, Boston.

It was voted to endorse Senate Bill 19, relating to the registration laws of Massachusetts.

Alephean Club

AT the regular monthly meeting of the Alephean Club held at 270 Commonwealth Ave., Boston, March 6, 1925, Dr. Cecil W. Clark of Newtonville presented a paper on Medical Legislation. The interest of the members present in this important subject was demonstrated by the active round table discussion which followed.

Harvard School of Public Health

THE Students' Club of the Harvard School of Public Health held its regular meeting at the School on March 18th, at 8 P. M.

Drs. Lubczynski, Nobechi and Huber were the speakers.

On April 2nd the members of the Club will meet with their guests for a social evening in the faculty room of the school.

Meeting of the Harvard Medical Society

THE regular meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital on Tuesday evening, March 10th.

Two cases were demonstrated. The first was a man of sixty-eight who had been injured in an automobile accident. He had extensive lacerations of the head and fracture of the patella. The second case was a woman of sixty years with pernicious anemia. In both cases X-rays were taken; in the first case to determine the extent and nature of the injuries and in the second to determine the impairment of gastric function in the condition of gastric anacidity. The interesting feature of both cases was that diagnoses of Paget's disease of the bones were made from accidental findings in the X-ray plates. Other evidence, confirmatory of these findings, was obtained from the physical examination and history such as decrease in stature, bowing of the legs and enlargement of the skull. Many cases of Paget's disease are probably overlooked and little is known of the possible relationship of this disease to other pathological conditions.

Dr. S. B. Wolbach addressed the meeting on the "Pathology of Deficiency Diseases." Dr. Wolbach and Dr. Percy Howe have been investigating the pathology of these conditions through animal experimentation. Very little is known of the pathology of deficiency diseases. A few of them can be reproduced in animals with a marked degree of similarity to the diseases in man. In monkeys a condition of the bones simulating Paget's disease can be produced as an end-result of scurvy or scorbutus.

Considerable work has been done on Beri-beri. This disease results from a lack of a "so-called" vitamine found in the outer layers of the kernel of grain. A closely analogous condition can be produced in fowls, and in a few hours they may be restored to normal by administration of the lacking substance. There is very little pathology to Beri-beri. About the only noticeable changes are a loss of chromatin and change in the nuclei of the nerve cells.

Rickets is most successfully reproduced in rats. The clinical manifestation of the disease is a softening of the bones. It is, in reality, an inability of the bones to harden. The condition

is not due to a lack of vitamines but to a disturbance of the mineral balance in the diet. When the calcium and phosphates are reduced to a minimum in the diet of a rat growth stops, but there are no signs of rickets. If the balance is disturbed by increasing one of these mineral substances, and not the other, rickets occurs.

In rickets, the intercellular substance is laid down normally in the bones, but the defect arises through failure of this intercellular substance to hold in chemical combination the necessary mineral constituents.

In their experimental work, Dr. Wolbach and Dr. Howe attempted to discover the earliest change that takes place with the feeding of a scorbutic diet. The first outward manifestation is lameness in the animal. Later there is hemorrhage about the mouth. At post-mortem, hemorrhage is found around the joints and beneath the periosteum. Histological studies show that the first changes occur in bone and cartilage, including the teeth. These parts cease to grow in a few days after feeding a scorbutic diet. The periosteal cells continue to proliferate, however, until about the fourteenth day, when a hemorrhage wipes them out. The cells multiply but produce no matrix. In the same way the cartilage cells and odontoblasts of the teeth close to form their characteristic intercellular substances. The changes are more extensive in localities subject to marked mechanical stress.

The striking observation of these experiments is that the animal shows a complete recovery within forty-eight hours after a return to an anti-scorbutic diet. Even in twenty-four hours there is a remarkable change. The odontoblasts, osteoblasts, etc., immediately begin to form intercellular substance again. This appears in such large quantities that it seems improbable that it is produced in the few hours following the change of diet. It would be more reasonable to believe that the anti-scorbutic has a jelling function, that the intercellular substance is already there in liquid form during scorbutus but cannot jell. Histological study supports this hypothesis. For example, in the teeth, the layer of odontoblasts seem to retract from the dentine during scorbutus, as though some substance occupied the intervening space. Upon recovery, this wide space is found to be filled with newly-formed dentine. Other intercellular substances are more or less affected in the same way.

Xerophthalmia may also be produced experimentally. This condition arises from the lack of a fat soluble vitamine.

In the autopsy of an infant with this disease, striking changes were found in the epithelium of the glandular organs. These changes primarily involved the ducts but, as it progressed, the gland structure also became affected. The cells are altered from a gland type to an epidermoid type. The normal epithelium is replaced by a stratified squamus epithelium, having no chemical function. Such changes are found in the lachrymal glands, the pancreas, the salivary glands, the glands of the bronchi, and also in others to a less extent.

The exact duplicate of these conditions was produced in rats. The most marked changes occurred in the lachrymal glands, epithelium of the renal pelvis, etc. In the ductless glands, such as the thyroid, the same changes did not occur, but marked atrophy of these organs was found.

Dr. Wolbach showed a number of lantern slides illustrating the striking histological changes that occur in these deficiency diseases.

The New England Heart Association

A MEETING of this association was held at the amphitheatre of the Peter Bent Brigham Hospital, Thursday, March 19, 1925, at 8:15.

Subject:—"A Clinical Study of 203 Cases of Malformations of the Heart." By Dr. Paul Emerson and Dr. Hyman Green.

Wachusett Medical Improvement Society

THE regular monthly meeting of the Wachusett Medical Improvement Society was held at the Holden District Hospital on March 4th. The paper of the evening was given by Dr. E. L. Hunt of Worcester City Hospital, Standardizing Stomach Ulcer Surgery. He mentioned the beneficial results obtained by medical treatment of some cases as against the results obtained by some surgical cases. For surgical cases, he felt that it was very essential that there be no residual in the stomach previous to operation. For twelve hours before operating, he allows no food and the last six hours not even water. He advocates stomach washing as a valuable aid in the preliminary training of some stomach cases. The paper was fully illustrated with lantern slides and was freely discussed.

Dr. E. B. Emerson of the Rutland State Sanatorium reported on the course in Tuberculosis which is to be given by the Department of Public Health in coöperation with the Wachusett Medical Improvement Society, the dates being May 6, 13 and 20 at 3 P. M. The program is: May 6, History, Dr. M. H. Joress: Physical Signs, Dr. E. B. Emerson; X-ray, Dr. W. B. Davidson; May 13, Laboratory Methods, Dr. Joseph Mueller; Juvenile Tuberculosis, Dr. H. D. Chadwick of Westfield State Sanatorium; May 20, General Management, Dr. E. B. Emerson; Treatment of Symptoms, Dr. H. C. Hubbard; Artificial Pneumothorax, Dr. W. B. Davidson.

The course is open to all who are interested and each subject will be illustrated by clinical demonstrations. Those enrolling are urged to bring their stethoscopes.

Enrollment and further details may be se-

cured from Dr. E. B. Emerson, Rutland State Sanatorium, Rutland, Mass.

O. DRAPER PHELPS, *Secretary.*

Hampshire District Medical Society

A REGULAR meeting of the Society was held at Forbes Library, Northampton, on Wednesday, March 11, 1925, at 11:30 A. M. Dr. George W. Rawson of Amherst presiding. A committee was chosen to draw suitable resolutions on the recent death of Dr. Paul A. Hudnut. Dr. Francis Lowell Burnett of Boston read a paper on "The Nourishment of the Body" which was well received. Dr. Burnett described the physiology of intestinal digestion in animals and man, outlining the benefits of a properly balanced diet, and illustrating his lecture by graphic charts. A general discussion followed.

SOCIETY MEETINGS

Essex North District Medical Society

May 6, 1925. Annual meeting at Lawrence.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held on the second Tuesday of March and May.

Hampden District Medical Society

Meeting to be held on the third Tuesday in April.

Hampshire District Medical Society

The meetings will be held the second Wednesday of March and May.

Middlesex East District Medical Society

Wednesday, April 15. Harvard Club.
Wednesday, May 13. Colonial Inn, North Reading.

Middlesex North District Medical Society

April 29, 1925.

Middlesex South District Medical Society

Winter Schedule—The plans for winter meetings of the Society include the stated meeting in April, two hospital meetings, and five meetings to be held in conjunction with the Suffolk District Medical Society and the Boston Medical Library (two surgical, two medical, and one general).

Norfolk District Medical Society

March 31, 1925. Tufts College Medical School. This meeting given over to Drs. Leary and Watters for the purpose of giving us a medical examiners' talk.

Norfolk South District Medical Society

Meetings will be held the first Thursday of each month to May, inclusive, at 12 noon, at the Norfolk County Hospital, South Braintree.

Suffolk District Medical Society

March 25. Medical Section, in association with the Middlesex South District Medical Society. "The Treatment of Pneumonia," Dr. Edwin A. Locke.
April 29. Annual meeting. "Hypertension and Longevity," Dr. Harold M. Frost.

Worcester District Medical Society

April 9, 1925. Subject and speaker to be announced.
May 14, 1925. Annual meeting.

If you desire further information in regard to these meetings write to the Secretaries of the District Medical Societies (listed on page viii of the Advertising Section). The Massachusetts Medical Society Directory contains their addresses.

BOOK REVIEWS

ACKNOWLEDGMENT OF BOOKS RECEIVED FOR REVIEW

Modern Aspects of Syphilis. By M. J. Horgan. London: Henry Frowde and Hodder & Stoughton. 136 pages. Price, $1.75.

Gynaecology with Obstetrics. By John S. Fairbairn. London: Humphrey Milford—Oxford University Press. 769 pages. Price, $8.

The Treatment of Fractures in General Practice. By C. Max Page and W. Rowley Bristow. London: Henry Frowde and Hodder & Stoughton. 239 pages. Price, $4.

The Theory and Practice of the Steinach Operation. By Peter Schmidt. London: William Heinemann. 150 pages. Price, 7/6 net.

The Baby's First Two Years. By Richard M. Smith and Mrs. Henry Copley Greene. Boston and New York: Houghton, Mifflin Company. 159 pages. Price, $1.75.

Crinnerungen Gedanten und Meinungen. By B. Raunhn. Munchen: J. F. Bergmann. 571 pages.

A Textbook of Physiology: For Medical Students and Physicians. By WILLIAM H. HOWELL, PH.D., M.D. Ninth edition, thoroughly revised. Octavo of 1069 pages, 308 illustrations. Philadelphia and London: W. B. Saunders Company. 1924. Cloth, $6.50.

Professor Howell's textbook of physiology, now in its ninth edition, needs no introduction to the medical profession. It gives a very readable account of physiological phenomena which is most welcome to the student, who is often bewildered by the more encyclopedic type of text or the special monograph. It is well balanced in that it touches briefly upon nearly every branch of the science, and as a rule the treatment has been brought well up to date. The sections on the chemistry of muscular activity and on the glands of internal secretion have been completely rewritten. The old arrangement of subject matter has been followed, and in certain cases this leads to unfortunate divisions, such as the separation of the neutrality regulation of the blood from the transport of carbon dioxide and oxygen. In contrast to the excellence of many chapters, notably those dealing with the blood and the special senses, the sections on nerve-muscle physiology (aside from that on the chemistry of muscle) seem rather out of date and unsatisfactory. The principle of "all-or-none" activity is scarcely mentioned, and there is a confusing use of the term "irritability."

The book is written primarily from the descriptive point of view, and tends to avoid controversial points in the interpretation of observed phenomena and "the danger that error rather than truth may result from the premature use of exact methods in the handling of confessedly incomplete data."

International Clinics. Volume II, 1924. 295 pages. Philadelphia and London: J. B. Lippincott Company.

In the symposium on physiotherapy which appears in this number there is an excellent article by Dorland on "The Influence of X-rays and Allied Substances on Living Tissues." Dorland reviews the literature on the effects of radiation on all forms of living organisms from the ameba to the human being. A very complete bibliography is given.

Tousey has written on certain phases of radium treatment. No new ideas are presented. His article is well illustrated.

Haines, Murray and Faber review the principles of caudal anesthesia. They point out that the sacral hiatus over the canal lies at one angle of an equilateral triangle, the two posterior superior spines forming the other angles.

The recent advances in surgery are discussed by Balfour and Flynn. The outstanding features are: The use of ethylene as a general anesthetic, the development of fluoroscopy in renal operations, the use of Lugol's solution in exophthalmic goiter, the gradual emptying of a distended bladder by a manometer control as suggested by Bumpus and Foulds, the intravenous use of tetrachlorphenolphthalein as an aid in the X-ray diagnosis of gall bladder diseases, the use of the living suture in hernia operations, and the experimental work of Royle and Hunter.

Ashhurst has written a well-illustrated article on osteomyelitis.

Pepper reviews the essentials of metastasis, of tumors, bacteria, filterable virus, bacteria and their toxic products, and inorganic elements.